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VOLUME 4 OCTOBER, 1926 NUMBER 4

ABLATION EXPERIMENTS ON THE LABYRINTH OF FROGS*

JOHN TAIT, M.D.

Professor of Physiology and Director of Experimental Medicine, McGill University. Illustrated with experiments by Dr. W. J. McNally.

MONTREAL

Mr. Chairman, Ladies and Gentlemen.—I wish first of all to express my deep appreciation of the honor of being invited to address these two important Societies, namely, the Otological and the "Triolog," an honor which for me is considerably enhanced by the fact of platform association with Professor Magnus, to whom and to whose fundamental work on the labyrinth we all take off our hat. In his charming "Tanglewood Tales," Nathaniel Hawthorne has retold the story of Theseus, who, sailing from Athens to Crete, slew the Minotaur in the great adventure of the Labyrinth. In view of his mastery over the intricacies of the labyrinth of the ear, we might well acclaim Professor Magnus as our Theseus. You must have felt when you read the title of my particular address that you had come to a physiology department with a vengeance. If you want to see a physiologist really happy, give him a frog. If, however, you want to see an audience really bored, give them a physiologist to speak to them about a frog!

After all, the labyrinth is common to the whole group of vertebrates —fishes, amphibians, reptiles, birds and mammals, from the lowest egg-laying platypus to the lord of creation himself. So that froggy is not in bad company. In the district in which I spent my youth we had a local poet, who once astounded his friends by composing a poem on the liver. Someone asked what possessed him to write on such a subject, and he replied: "Ah! I searched through the whole of the English literature, and found that the great poets had written on all the organs of the human body except the liver; that is why I wrote my poem." It is noteworthy that physiologists, who have searched through and written on all the organs of the body of the frog, have shown a marked disinclination to deal with its labyrinth, and for a very good reason. The frog's labyrinth is of such tiny dimensions and is withal so delicate, that had it not been for the conspicuous ability of Dr. McNally in carrying out the finest differential operations on it, I should not have

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Read before the Joint Meeting of the American Otological Society and the American Laryngological, Rhinological and Otological Society, Inc., June 2, 1926, at McGill University, Montreal, Canada.

been talking to you today. Once these operative difficulties had been overcome, the frog proved to be a particularly valuable animal—superior in many respects to the customary fishes and birds—from which to deduce conclusions regarding the function of individual parts of the labyrinth.

In the labyrinth of the frog there are the usual parts—the saccular macula, the utricular macula and the three semicircular canals. Dr. McNally performed operations on each of these parts, and I shall speak of them in succession. There is one point, however, which I must make quite clear: We did not pay any attention to the eyes or eye movements of the frog. Some of you, I have no doubt, will be relieved to hear this; others will be correspondingly depressed. My only hope is that the disappointment of the one party will be balanced by the satisfaction of the other. Our observations on disturbances of equilibrium concern only the limbs and body of the animal.

THE SACCULAR MACULA

The illustration on the screen represents the saccular otolith and macula of the frog as displayed in a dissection by Dr. McNally. When one, or both, saccular otolith is removed, or, what is still more certain and conclusive, when the nerves to the saccular maculae are cut, the result so far as equilibrium is concerned is very clear and definite. The animal sits, crawls, jumps and swims absolutely in normal fashion. After any operation whatsoever on the saccular maculae we could not detect the slightest variation from normal behavior, and the animals were elaborately tested. These wholly negative results are simply a confirmation of previous work both on fishes (by C. H. Parker and by S. S. Maxwell) and on frogs (by Laudenbach), which had gone to show that the saccular macula, at least of these lower forms is not in any way concerned with equilibrium.

THE UTRICULAR MACULA

I do not wish to dwell much longer on the utricular than on the saccular macula. I have no right to spend time on it, first, because we have nothing new to report, and second, because our operations on the utricular macula were in the end entirely accidental. The utricular macula happens to be particularly difficult of access and exposure, and we have no lantern illustrations even of the preserved and dissected organ to show. The nerve to the utricular macula is distributed very close to the two anterior ampullae of the semicircular canals, and either it or the macula itself is exceedingly apt to incur injury during operations on the canal ampullae. Any such accidental injury, however, even though uncontrolled, is accompanied by uniquely characteristic symptoms, which show with the greatest clearness over what kind of reaction the utricular macula presides. The utricular macula is wholly an organ

of static equilibrium, as has been recognized since the time of Breuer and substantiated very definitely by Professor Magnus and his school. I emphasize this, because there are still hesitant statements in the literature as to the respective parts played by the canals and by the utricular macula.

It is necessary to be quite clear as to what we mean by static equilibrium, and this involves a short but vital digression. On the table before me I have a rigid model, constructed of three straight rods set at right angles to each other and intersecting at one common point. One rod points vertically; it represents the direction of gravity. The other two lie in the horizontal plane, one running north and south, the other east and west. The model represents the physicist's or mathematician's axes of reference in specifying displacements of a body. Now, if I take this oblong piece of board (on which we might allow a toy animal to stand with its body axis in the line of the board) and place it transversely across the N-S axis, both board and animal are still in the horizontal plane. So soon, however, as I rotate the board ever so slightly about this axis, the animal becomes tilted, either head downward or tail downward, as the case may be. If, without changing the direction of the (horizontal) board, I transfer it to the E-W axis and rotate about this axis, the animal is tilted either rightside down or leftside down, as the case may be. In other words, any rotation about a horizontal axis, whether this axis be N-S or E-W, tilts the animal out of the horizontal plane, the consequence being that it is apt to lose its balance and topple off. Yet see what happens if I cause the board to rotate slowly about the vertical axis! It remains horizontal, pointing now N-S or E-W or in any intermediate direction. Meantime the animal on the board also remains horizontal with no disturbance to its equilibrium whatsoever. There is therefore one axis, namely, the vertical, rotation about which is of indifference so far as gravity is concerned. Rotation about this axis causes no tilting and no tendency on the part of the animal to topple over. When we come to think of it, we use the word "tilt" solely in reference to rotation about some horizontal axis. Only when the subjacent plane surface becomes tilted does the animal incur danger of upset.

When this surface is inclined or tilted out of the horizontal, the living, unlike the toy, animal reacts and adjusts its posture so as to be in stable equilibrium in spite of the new inclination of the surface. This reaction is purely a reaction against gravity, and the response is automatic, occurring just as well in the decerebrate as in the intact frog. The body of the animal becomes so placed as to rest vertically above its four limbs. The head, too, tends to be held horizontal.

(Lantern slides were here shown, representing stationary postures assumed by the frog on an inclined plane. In each case the head tends to be kept horizontal, while the center of gravity of the body is plumbed within the base of support formed by the four limbs.)

These stationary postures, which vary with the degree of inclination of the plane on which the frog rests, constitute reactions of static equilibrium. In taking up the appropriate pose the animal depends on nervous messages derived from four possible sources: (1) Pressure impulses due to contact with the ground; (2) impulses from muscles and joints; (3) impulses from the eyes; (4) impulses from the utricular maculae. That these last impulses are of outstanding importance can be proved by the result of injury to the utricular nerves. When, in an otherwise almost intact animal, the macula is denervated on one side, the frog, notwithstanding the nervous messages from its eyes, from its muscles and from its palms and soles in contact with the ground, no longer squats naturally. On a horizontal surface it leans over to the operated side, its head and body having much the same relation to this surface as if the animal were intact and the surface had been tipped downward on the nonoperated side. If both utricular maculae are denervated, the animal, when laid on its back, has great difficulty in righting itself. If it is blindfolded and thrown into water, it loses all sense of the direction of gravity. This is why human patients with bilateral defect of the utricular maculae risk their lives if they dive into muddy water. When a mason or a carpenter wishes to place some structure in a vertical position, he uses a plumb line. In the information they convey respecting the direction of gravity, the utricular maculae may be roughly compared with the plumb line of the mason.

If we now glance backward for a moment, we should make these statements with regard to the two otolithic organs of the labyrinth. So far as the body and limbs of the frog are concerned, the saccular macula has nothing to do with equilibrium. The utricular macula is the organ of static equilibrium, and is concerned in adopting and maintaining appropriate postures in response to gravity. Before going further, let me also repeat that any rotational shift of the horizontal substratum, whereby it remains throughout horizontal, is entirely neutral as regards these static postures. Only when the horizontal plane becomes tilted is a new posture assumed.

THE SEMICIRCULAR CANALS

The semicircular canals—and it is to them that I wish to devote the main part of this lecture—are for kinetic equilibrium. Breuer made the great distinction between static and what he called dynamic equilibrium. I am inclined to prefer the word "kinetic" to "dynamic," because in the end even static or simple gravity reactions are dynamic. But a mere difference of terminology is neither here nor there.

What exactly do we mean by kinetic as opposed to static equilibrium? If we consider plants as well as animals, we can readily understand the difference between the two. A plant or a tree grows vertically

upward in opposition to the force of gravity. Whether it is rooted on a plain or on the side of a sloping bank it uplifts and holds its stem in the vertical direction. In other words, it shows static equilibrium. Animals by constrast are motile creatures that run and skip about on legs. They turn and twist and yet maintain their balance. They frisk and jump, and, after springing into the air, land adroitly on their feet. These accomplishments are reactions of kinetic equilibrium, the static reactions being manifested only when the animal stands still.

How do we proceed to test an animal for disturbance of kinetic equilibrium? If kinetic equilibrium is involved only during movement, it might seem at first sight a hard matter to make accurate observations. Yet the testing process is easy. The animal is allowed to sit still. All that is necessary is to jerk it about in an effort to upset it suddenly. A normal animal by prompt counter-reaction tends to maintain its balance. A decanalized animal is far less quick and is much more readily capsized.

On a number of occasions Dr. McNally succeeded in removing all six semicircular canals from a frog, without other damage to the labyrinths. We had hoped to show you one of these animals, and it is not his fault in any way that we cannot do so. This is the worst possible period of the year in which to operate on frogs. Just after the spawning season they succumb to operation and to sepsis much more readily than they do in winter. I have, however, a model here with which to show you the kind of effect. It consists of a piece of wood to represent the body of the animal. This is mounted on four spindly wire legs fixed into a wood base, like a toy horse. When I now weight the body with a piece of metal the sham animal still stands upright, and to this extent shows static equilibrium. When, however, I place the weighted model on a horizontal tilt-table and sharply tip the table through a small angle, the heavy body sways and rocks about. It is obviously wobbly on its legs, and that is exactly the impression given by a completely decanalized frog when subjected to some sudden displacement. A normal frog, as Dr. McNally will now show, behaves quite differently. [Demonstration.] No matter how quickly it is tilted from the horizontal, its muscles are immediately innervated and it reacts so as to withstand the displacement. There is a promptness of reaction in the normal frog which is altogether absent in the decanalized animal.

Over its static equilibrium the decanalized frog has complete control. Set it on a tilt-table and incline the table ever so slowly. To each new angle of inclination it adjusts itself beautifully so that its body remains vertically over the base of support and its head is held horizontal. Its plumb line mechanism is therefore working. But you must be careful at every stage to go slowly, for the utricular maculae and their connections are more deliberate in operation than the canals. So soon

as we proceed to tilt quickly, the reflex static mechanism is found to be altogether too tardy, and the animal, lurching to the downward side, may lose its stability and topple off the table. Professor Magnus has already referred in his lecture to this interesting feature of canal function brought out in our experiments, whereby the intact animal, subjected to some sudden tilt, is on the cessation of the tilting movement already in the position that it is going to maintain under the uncomplicated influence of gravity.

This then is the first great characteristic of semicircular function, namely, that appropriate responses are made—quick, rapid, almost lightning-like—so as immediately to counteract sudden tiltings and jerkings of whatsoever kind. The canals are called into action by something sudden; their effect is momentary and evanescent. The plumb line mechanism responds to a steady field of force; its action is sluggish but its effect is sustained. This is somewhat reminiscent of the difference between an induced and a constant electric current.

We now perhaps have a better conception of what is involved in canal function and kinetic equilibrium.

Differential Function of the Canals.—As you know, there are six semicircular canals, three on each side, and it was one of Dr. McNally's achievements to effect clean and uncomplicated ablations of any single canal ampulla or of any combination of them. As a result, we obtained a deeper insight into the function of the individual canals. As an experimental animal on which to determine the effect of these ablations, the frog proved to have quite peculiar qualifications. Let me stop for a moment, however, to explain how the canals sit in the head.

Each canal is a curved tube with a dilatation or ampulla at the end of it, in which in each case is situated the sensory organ and to which the sensory nerve is distributed. I have on the wall a classical diagram by J. R. Ewald, representing the position of the canals in the head of the pigeon. By painting the canals, as it were, on transparent sheets of glass, he has well represented the planes in which they lie. The matter can be made equally clear without a diagram, provided one uses one's arms to represent the canals, taking them in pairs at a time; by this method, too, the arrangement becomes more easy to remember. Let my bent arm represent the tube of the canal and let my closed fist represent the ampulla. Then if, curving both arms, I hold them out on a level in front of me, the fists about two feet apart, I am showing you the position of the horizontal canals, the ampullae of which, you will note, lie anteriorly. As the name implies, the horizontal canals lie in the horizontal plane.

Let me now illustrate the position of the two anterior vertical canals. Holding my arms out as before, I swing my elbows upward. My fists, which are the ampullae of these canals, remain near where they were before, for the ampullae of the anterior vertical canals are close to those

of the horizontal canals. The tubes of the vertical canals lie in vertical planes, and note how these tubes point. Not straight forward and parallel to each other, but forward and laterally in each case. Now if I swing my curved arms behind my back, keeping the curves, so far as may be, vertical, my fists, which in this case represent the ampullae of the posterior vertical canals, point backward and also laterally. In other words, the four vertical canals, lying in vertical planes, point toward the four corners of the body, and in each case the ampulla is at the outer extremity.

Differential diagnosis of lesions of the canals depends of course on a knowledge of their individual function. At the present time there is one outstanding method, which we owe to Bárány. By an ingenious combination of his caloric test with rotation of the patient on his revolving chair, Bárány showed how it is possible to infer destruction or malfunction of particular canals. His caloric test, as you know, depends on unequal distribution of temperature within the labyrinth. He cools the external ear passage and so produces a localized cold region of the bone inclosing the labyrinth. Meantime, this bone is kept warm by the steady flow of blood from the internal carotid artery, which passes up like a hot stove-pipe beside it. The utricle is similar to a boiler, with which the tubes of the semicircular canals are connected, and, just as in a hot water system a circulation can be established in the pipes connected with the boiler, the warm fluid mounting, the cold fluid descending, so Bárány was able, by turning the head now into this position, now into that, to bring any canal for the time being into a vertical position and thus to stimulate it if normal and healthy. His method provides a beautiful proof that stimulation of the canals is due to movement of the endolymph within them, but in actual practice it is complicated and not wholly satisfactory. For one thing, he makes his observations on the eyes of the patient, and it takes experience to observe these with precision. For another thing—and Professor Magnus has dealt with this today—mere bending of the neck tends of itself to cause eye movements. In our work we left the eyes out of account. We found that by observation of limb movements it is possible, at least in the case of the frog, to diagnose quite simply an individual lesion of any of the four vertical canals. The method is capable of easy extension to human beings.

THE HORIZONTAL CANALS

These are brought into action in rapid turning movements about a vertical axis.

Let me go back once more to this model of three rectilinear axes at right angles to each other. You remember that when an animal is rotated in the horizontal plane, that is, about a vertical axis, its static posture remains unaltered, for whether it points from north to south or from

east to west, gravity acts on it in similar fashion in each case. It is only when rotation occurs about a horizontal axis, in other words, when the horizontal plane itself is tilted, that new static postures are necessary. Why, then should there be a pair of canals which respond to turning movements about a vertical axis?

Let me illustrate with a rather crude model. Here I have an animal with a body shaped somewhat like an hour-glass, a kind of hybrid between a pig and a wasp. The body is made of a cylindric cotton bag like a miniature pillowslip. Into the front end, 4 inches of sand was poured and a ligature tied closely behind the sand. A second ligature was tied 2 or 3 inches farther back, then some more sand was introduced to represent the hinder part of the body, while a third ligature closed the end. The result is that there is a kind of swivel or flexible portion in the middle of the body. By means of cement the fore part is mounted on a piece of wood, which may represent the forelegs; the hinder part is mounted on a similar piece of wood to represent the hindlegs. I place the animal on this horizontal turn-table, with the middle or flexible portion of its body over the vertical axis. If I turn the table gently the animal remains in stable equilibrium; it makes no difference whether it heads toward the south or toward the east; and when I stop turning, it stays as before. All this we have already discussed. Provided the turning is slow, the animal remains in secure equilibrium. But see what occurs when I grasp the spindle of the turn-table and make a quick turn of a quarter circle in the direction of the sun. The animal falls, and observe what has happened. The fore part of its body has fallen over to the left side, the hind part has fallen over to its right! That is why the swivel was introduced into its make-up—in order to permit torsion of the trunk. I repeat the experiment that you may see it again. You will now be prepared to admit that it makes quite a difference whether turning, even about a vertical axis, occurs slowly or rapidly.

Note how the animal lies. Its forelegs point toward its right side, its hindlegs toward its left—and naturally so, because I pulled its forelegs from under it in one direction, and its hindlegs (on the opposite side of the vertical axis) in the other. If I set the animal up again, consider for a moment what it ought to do with its forelegs if it should attempt to counteract the quick turn of the table in the direction of the sun. Obviously, as the ground moves from under it toward the right, it should step sideways with its forelimbs to the left. How should it step with its hind limbs? Obviously, sideways to the right. But if it steps to the left with its fore-limbs and to the right with its hind-limbs, it is performing a circus movement in the direction opposite to that of the turning of the turn table. This is exactly what a real animal does when quickly rotated about a vertical axis to one or other side. It performs circus movements in the opposite direction to that of the turning,

and these circus movements contribute toward better momentary equilibrium. The canals which are stimulated in these quick turnings about a vertical axis are the horizontal canals.

Dr. McNally has two frogs here, one a normal animal, one a frog in which he has destroyed the ampulla of the right horizontal canal. As he places the first frog on the turn table and gives the spindle a quick, quarter-circle twist in the direction of the sun, the frog makes an equivalent circus movement in the opposite sense. When he quickly twists the axis of the turn table in the reverse direction, the frog as promptly moves against the direction of turning. When he turns the table quite slowly, the animal remains sitting still. The interesting experiment is now to come. How does the animal with the defect of the right horizontal canal behave? The table is twisted sharply in the direction against the sun, that is, toward the animal's left. The frog behaves normally and makes a short circus turn in the opposite direction. The table is now sharply turned toward the animal's right, but there is no reaction. This proves that when the normal frog is quickly rotated toward its right, it is the horizontal canal on that side that is stimulated. Had the lesion been effected, not on the right, but on the left side, the frog would have failed to respond when turned toward its left.

This result, so simple to remember, is not new. By operations on the horizontal canals of pigeons, Ewald long ago showed that rotation of the head to the right stimulates the right horizontal canal, and vice versa. At the same time, the frog experiments, being carried out on a wholly different class of vertebrates, give a wider basis to his deduction. As the conclusion holds true in respect both of birds and of amphibians, it may well apply also to mammals and man.

THE VERTICAL CANALS

From the previous exposition you will have already concluded that the vertical canals are brought into operation in rapid tilting movements about horizontal axes. Suppose I were asked to fix structures such as the canals on a steamer, in order to signal disturbances due to wave movement. If I placed one pair in the fore-and-aft direction, they might signal the pitching movements. A pair in the transverse direction would signal the rolling movements. Why are our vertical canals not placed in this fashion, which would seem to be so simple and natural? For one thing, you must remember that the canals are not placed in a central position in the head, but in two laterally disposed, symmetrical groups of three; this introduces a complication with which, as some of you know, Crum Brown signally wrestled in an acute paper of purely theoretic content. For another thing, we must keep in mind that an animal is no steamer. Land vertebrates walk on four struts or legs, and in the usual form of progression the body is supported alternately

on pairs, consisting of right fore and left hind, and of left fore and right hind respectively. In each case the girder bridging the upper end of the limbs passes diagonally across the trunk, and the body may be said to swing in each case about a diagonal axis. Now, with respect to the vertical canals, the right anterior and the left posterior can be considered as constituting a parallel pair, while the left anterior and right posterior constitute another parallel pair.

As the vertical canals are arranged not anteroposteriorly and transversely, but as two diagonal pairs, the best way to orient an animal on the tilt-table in examining for defect of the vertical canals, is diagonally. Suppose we wish to test the left anterior and the right posterior pair. The animal is placed in such a way that this pair of canals lies at right angles to the axis of rotation; meantime the other pair, lying parallel to the axis of rotation, will not be stimulated. In order to be more exact in our specification, let us call one end of the tilt-table the front and the other the back; think of it as being the top of a two-wheeled cart, with the shafts to indicate the front. I place a toy dog diagonally on the horizontal cart. His head points to the right front corner, his tail to the left back corner of the cart. When he is so placed, which pair of canals will be stimulated by tilting movements? Obviously the left anterior and right posterior. I slowly depress the shafts. Plainly, then, the weight mainly rests on the left fore-limb. Suppose the tilting occurs suddenly, what should the animal forthwith do if he is to preserve his equilibrium? He should render this limb very firm and rigid, or even throw it farther away from the axis of rotation, that is, farther in the direction of the left-hand corner of his body. It will then best act as a strut to prevent toppling. This is exactly what a living intact animal does under the circumstances.

By raising the shafts I bring the dog back to the horizontal position, and now I tip the back of the cart downward. His weight now falls mainly on his right hind-limb. Should the tipping movement occur suddenly, a real dog would immediately stiffen this limb, or then throw it farther backward and laterally, so as to meet the shock of the toppling body.

Now for Dr. McNally's demonstration of the effect of an actual experimental lesion. In the frog that he exhibits he has destroyed the ampulla of the right posterior vertical canal. He places the animal on the tilt-table exactly as I just placed my dog. The front end of the table is tipped quickly downward. The frog easily preserves its balance. It would seem that absence of a posterior canal is of no consequence in a case of forward tipping. Now the direction of tilting is to be reversed, and, please, observe closely. The animal, obviously taken unawares, rolls backward over its limp right hind-limb and falls off the table. The limb was not ready for its duty, which was to stiffen or extend and so

to resist capsizing of the body. The experiment can be varied. Instead of placing the frog diagonally, Dr. McNally now sets it in the fore-and-aft direction, that is, with its body transverse to the axis of rotation. He tips the table backward. The left hind-limb is shot out backward so as to save it against a fall, the right hind-limb fails to move. On repetition of the experiment with a still sharper rate of tilting, the frog actually falls over backward and to the right. You had just time to see that, whereas the left hind-limb was quickly and promptly extended, its partner on the right failed to move—whence the peculiar nature of the backward fall.

Numerous experiments of this kind, both on the anterior and on the posterior vertical canals, led us to the following interesting conclusion. Each vertical canal is especially associated with the limb of that quarter of the body toward which it points. A vertical canal is stimulated when the head is suddenly tipped downward toward the quarter to which the canal points. The main effect of stimulation of a vertical canal is a sudden extension of its own particular limb, which is thrown diagonally away from the body, that is, further toward its own corner. In this way the limb is prepared to exert a thrust (1) upward, against the weight of the falling body, (2) horizontally, in the direction toward the center of the body. We may also note that whereas a vertical canal is specially associated with one particular limb, stimulation of a horizontal canal leads to movements of at least two, and usually of all four limbs.

If we extend our survey over the animal kingdom, we find that semicircular canals are peculiar to vertebrates. An outstanding feature of vertebrate organization is the possession of four limbs. With these, the four vertical canals are especially associated. We first meet with semicircular canals in the lowest fishes, or cyclostomes; these have four vertical but no horizontal canals. Only when we come to the true fishes do we meet with horizontal canals. If we may argue from this, we should say that the set of vertical canals is the more primitive and the more essential. They guard and protect the land vertebrate against sudden falls due to stumbling or otherwise. When the land vertebrate eventually reaches the stage of raising himself up and walking on a single pair of limbs (compare birds and man), his equilibrium is more unstable, and the responsibility of the vertical canals is greater than ever. Yet their essential mode of function is the same in man as in a frog. We are walking along the road; our foot catches some obstacle; the body being momentarily held back, our head is smartly jerked downward and forward, just through the smallest angle. Before we have time to think our arms are shot out in front of us, and should we actually lose our balance, they are already in the proper position to save us. It is rare, however, during walking, to stumble straight forward. As a rule

one falls somewhat toward the side on which the foot first gets caught. In such a case it is invariably the arm on that side that is thrown outward, for the head has momentarily dipped toward that side. If we stumble backward and to some particular side, the head is made to jerk backward and to that side; as a result, the leg on that side is violently and forcibly thrown backward and outward. A forward stumbling bird, that is, a young chick, throws its wing forward and with the help of resistance from the air saves itself. Indeed, the very act of violent forward propulsion of the arm of a man during a stumble, by counter-reaction, tends to check the initial rate of fall of his body. The whole question of kinetic balance becomes of consuming interest so soon as we begin to appreciate the function of the vertical canals.

DIFFERENTIAL DIAGNOSIS OF CANAL LESIONS

We have seen that the canals fall into two groups, (1) what we might call a nongravity set, which is concerned with waltzing movements, during which the body remains steadily at the same uniform level, moving neither up nor down; (2) a gravity set, which is concerned in leaping and landing again on the ground, in protection against falls—generally speaking, in movements in which the limb muscles are forcibly exerted in opposition to gravity. For differential diagnosis of lesions in the human subject involving one or other of these two sets, we might reasonably use two different forms of apparatus, just as we did in the case of the frog, namely, (1) a Bárány chair, rotating about a vertical axis, wherewith to test the nongravity set; (2) some form of tilt-table, rotating about a horizontal axis, wherewith to test the gravity set. To meet the latter requirements, we have designed and constructed a special table, which you see here beside a Bárány chair. The two are complementary pieces of apparatus. They belong together but have separate uses.

Let me first of all make quite clear that we have carried out no elaborate simultaneous investigation with the two pieces of apparatus. Through the kindness of Dr. Birkett and of Miss Macleod, superintendent of the Mackay Institute for Deaf Mutes, we have had an opportunity of testing a limited number of deaf-mutes. During a recent visit to Cleveland, I was also privileged, thanks to the courtesy of Dr. Ingersoll and Dr. Kendall, to test one or two other cases. I shall merely explain how the tests were carried out, leaving them to your experienced criticism. With a young patient from the Mackay Institute, Dr. McNally will illustrate the mode of procedure, and you will be able to see for yourselves with what ease sudden tilting on the table brings out defect of the vertical canals.

First, with regard to the Bárány chair. The only test that we have used—and this is not the conventional method—is to set the patient on the chair and, after blindfolding him, to rotate the chair through a small

angle with varying degrees of angular acceleration. The patient is asked to indicate, by signing with the right hand or with the left hand, respectively, when he experiences rotation and the direction of this rotation. As Raymond Dodge has shown, an angular acceleration of 2 degrees per second suffices to cause conscious sense of rotation in a normal person. A rate of acceleration below this does not excite the horizontal canals, to keep below which threshold one must take care to speed up very slowly indeed. Somewhere in the cerebral cortex there must be a region for conscious experience of rotation, including discrimination of the angular sign of that rotation. In Head's elaborate investigation on sensory localization in patients who had incurred brain injuries during the war, their reaction to rotation was not investigated, unfortunately. It would be interesting to know where the center is.

In our limited experience, most deaf mutes on the Bárány chair have no trouble in detecting even a low rate of angular acceleration, and in correctly indicating its direction. Occasionally, however, one finds a patient who shows obvious disability. His threshold is high; his answers are incorrect. We have examined two such patients, and both had acquired bilateral labyrinth trouble as a result of meningitis. These two patients, when tested on the tilt-table, showed behavior analogous to that of a completely decanalized frog. The other deaf mutes that we examined reacted on the tilt-table the same as normal persons. Thus you will see that we have had no opportunity of making differential observations on single lesions of individual canals. The point of interest is that when we found a patient defective under the very simple test with the Bárány chair as just described, this same patient showed disability on the tilt-table.

Dr. McNally will now demonstrate the use of the tilt-table. As you will see, it has a broad, square top. The patient is placed on it resting on his hands and knees, and the breadth is to permit of his taking up any orientation, whether parallel to the sides or diagonal. A stout handle attached at one end—like the shafts of a cart—gives leverage and protects against any sudden tipping of the unstable platform.[1]

First of all, we demonstrate the behavior of a normal person. As he goes down on his knees he places his hands flat on the top of the table; he is not allowed to grip the sides. It matters not in which direction he is placed, anteroposterior, transverse or diagonal, his kinetic equilibrium during sudden jerks and throws of the table is perfect. [Demonstration.] Provided the angle of inclination is not made too steep, he is like a bird on a swaying branch and cannot be dislodged. The curious thing is that it costs him no effort and no attention. His balance

1. The table will be described and illustrated in a separate communication. It is made by the mechanic to the Department of Physiology and Experimental Medicine, McGill University.

is maintained by a wholly automatic process, and you will notice how his body immediately and invariably moves in a direction contrary to that of the tipping table. If any member of the audience imagines that this subject has had special training in tight-rope feats, let him come down and be tested himself at the end of the lecture. It always gives a mild shock of surprise to discover with what ease and with what insouciance the appropriate movements are made. The thing simply happens, and the individual as such can take no credit for his unsuspected endowment. To one who might not care to be reminded of his quadruped ancestry, it is illuminating once in a way to experience the equilibrial unconcern of a dog or of an ape.

Let us now observe under similar conditions the patient who has suffered total loss of her canals. She takes her place on hands and knees, this time diagonally across the table, for it is in this position that we examine a particular pair of vertical canals. Were she to slew round through 90 degrees and occupy the other diagonal, we should test the other pair of vertical canals. It does not matter how she holds her head, upright or looking downward, provided merely that it is in line with the body. At each end of the table a guard is stationed beside her in case of a fall. [Demonstration.] You can see the marked difference between the patient and a normal subject. When she is suddenly tilted forward or backward, even through an angle of, say, 10 or 15 degrees, she is upset just as easily as if she rested on paralyzed limbs. Note, too, the swaying of the body even on the slightest tilt, and think of the model on spindly wire legs that I showed you some time ago. The resemblance, I think, is plain. You will understand why, in testing a patient, he should be asked not to grip the sides of the table. You can see that the patient is anxious under test. If left to follow her own devices, she would hold fast to the table edge and stiffen her arms in the hope thereby of better resisting any unexpected movement. Indeed such patients prefer if possible to hold on rigidly, and, like a man clinging to the swaying mast of a ship, to become for the time being a part of the moving system. Could anything better illustrate the beautiful responsiveness of the muscular mechanism under canal control than a comparative demonstration of this kind, the canals being intact in the first subject and obliterated in the second?

I might mention that this young patient also suffers from utricular disorder, but that has nothing to do with her helplessness on the rapidly tilted table. If we ask her to stand on the table, facing in the direction of the axis, her feet somewhat apart, she can keep her balance during slow tilting, provided her eyes are open. If she is blindfolded she topples over when slowly tilted, whereas a normal person under similar circumstances keeps his balance.

CONCLUSION

With this demonstration I conclude. We began with the frog, and we ended with the human being. I do not say that the labyrinth of the frog reacts in every way like our own labyrinth, or that experimental results obtained with this lowly vertebrate form can be forthwith applied to ourselves. One thing, however, you may be ready to grant, namely, that a case has been established for routine examination of patients with labyrinthine disorder by the double method of the Bárány chair and of the tilt-table. When further observations have accumulated, we shall really be in a position to decide whether lesions of individual canals in the human being can by these means be as readily and as certainly diagnosed as they can in the frog.

POSTADENOIDAL SPEECH

JOHN A. GLASSBURG, M.D.

Laryngologist to the New York City Children's Hospital, St. Joseph's Hospital and the Stuyvesant Polyclinic; Medical Director of Speech Reeducation, Greenwich House

NEW YORK

Temporary and permanent speech disorders occur after tonsil and adenoid operations. It is not uncommon after tonsillectomy and adenoidectomy to find that the patient regurgitates liquids through the nose, and that his speech has a peculiar nasality. The sounds that are ordinarily resonated in the oral cavity only are also resonated in the nose.

Usually the condition clears up spontaneously in a week or two. When such is the case the trouble was due to impaired activity of the velum. In those patients in whom the difficulty persists, the cause is failure on the part of the soft palate and the uvula to occlude the nasopharyngeal space when the nasal sounds n, ng and m are phonated.

The faulty closure is caused by the removal of the adenoids. The nasopharyngeal space devoid of the masses of adenoids is too wide for the soft palate and the uvula to bridge across. As a result, the air rushes up through the posterior nares and there is an increased nasal resonance with an unpleasant, muffled voice production.

COMPENSATORY NASOPHARYNGEAL WIDENING

Normally, the uvula and soft palate are sufficiently large to close up the nasopharyngeal space, but when huge adenoids are present for a long time, the nasopharyngeal space becomes widened to accommodate the adenoid masses and at the same time to permit the passage of some air. This may be considered as a compensatory widening of the nasopharyngeal space.

When the adenoids are removed, the uvula and soft palate are insufficient in size to span across the space, and instead of too little air through the nose there is a gush of too much air.

HYPERNASALITY

In most cases, after the removal of the adenoids the compensatory widening of the nasopharyngeal space gradually comes down, and the soft palate and uvula by increased exertion and practice learn to occlude the space. In those cases, when due to either too great a widening or too short a soft palate and uvula, the failure to close up the space remains and a speech disorder develops, the chief characteristic of which is hypernasality.

The existence of a true hypernasality can be determined in several ways. In listening to the speech it will be found that sounds other than n, ng, and m are nasalized. If the patient is told to repeat a test sentence free of nasal sounds, it will be found that the speech is muffled and many sound tones are impregnated with the n element.

To make certain that the hypernasality is due to a short velum, the patient is told to tilt his head back and repeat slowly with emphasis on the A, the word RAH. The velum should rise and occlude the post-pharyngeal space on the A.

TREATMENT

The treatment consists of electricity and massage, which are more theoretical than practical, and special exercises directed to the uvula, palate, tongue and lips. As a prophylactic measure, every patient with an operation for adenoids in prospect should have the palate inspected to determine any unusual shortness.

TONGUE EXERCISES

1. Push the tongue out of the mouth and downward toward the chin.
2. Push the tongue outward and upward as far as possible, even touching the nose.
3. Point the tip of the tongue upward and downward alternately for a count of 10.
4. Rotate the tongue to the right, then down, then to the left, then up.
5. Protrude the tongue, grooving it at the same time by raising the sides.
6. With the mouth kept open, curl the tip of the tongue back of the front teeth.
7. Press the tip of the tongue to the back of the front lower teeth; then relax.

LIP EXERCISES

1. Pucker the lips and protrude them.
2. Raise the upper lip, exposing the upper teeth.
3. Wrinkle the nose so as to expose the upper teeth.
4. Raise the right side of the upper lip.
5. Raise the left side of the upper lip.
6. Pull down the lower lip, exposing the lower teeth.

SOFT PALATE AND UVULA EXERCISES

1. With mouth wide open, say ah-ah, and watch the palate rise.
2. Yawn and watch the palate rise.

365 West End Avenue.

TYPHOID FEVER SIMULATING SIGMOID SINUS THROMBOSIS

O. JASON DIXON, M.D.
KANSAS CITY, MO.

Such remarkable progress has been made in preventive medicine since the tragic days of Osler [1] at the opening of Johns Hopkins Hospital, that we are inclined to forget that such a disease as typhoid still exists.

Mosher,[2] in a recent discussion on the differential diagnosis of lateral sinus thrombosis, sharply condemned the worn out confusion with typhoid fever.

Having at the time just gone through the trying ordeal of differentiating typhoid fever from a septic thrombosis of the sigmoid sinus, I was deeply impressed with his remarks. In the management of this case it required considerable courage to withstand the onslaughts of the pediatricians, who flatly refused to accept any responsibility unless the mastoid infection was properly cared for. The history and rather unusual course is as follows:

REPORT OF CASE

F. Y., a boy, aged 10 years, was readmitted to Mercy Hospital, Oct. 5, 1925, on account of an earache on the right, and a temperature of 102 F. Ten days prior to this, I had removed the patient's tonsils and adenoids. The postoperative course had been uneventful.

Physical examination was negative, except for a ruptured bleb overlying the right red eardrum. There was no mastoid tenderness; the boy appeared drowsy, said that his back ached and that he felt tired. Two days before he had had a profuse epistaxis. The urinalysis was normal and the white blood count was 8,300.

Twelve hours following admission the temperature rose to 104 F. and remained between 102 and 104 for three days, when it fell to 99.2. Following this, he had a severe chill lasting for fifteen minutes and a sharp elevation of temperature to 105.3. The white blood count was 5,200.

Exactly twenty-four hours later the temperature again dropped to 99, he had another severe chill, an elevation of temperature to 105, and the leukocyte count was 4,600. The patient did not complain of pain at any time. He slept well, took fluids freely and appeared drowsy and listless. The right eardrum regained its normal luster. The temperature stayed between 101 and 106 for two days, then fell to 100, and returned quickly to 105, without a chill. The white blood count was 4,300. The pulse varied between 90 and 110 and the respiratory rate was 30. The urine remained normal, and the bowels were constipated.

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1. Cushing, Harvey: The Life of Sir William Osler, New York, Oxford University Press, Vol. I, p. 311.

2. Mosher, H. P.: From the discussion of a paper by Tobey on The Differential Diagnosis of Lateral Sinus Thrombosis, at the Academy of Ophthalmology and Otolaryngology, Chicago, October, 1925.

Blood smears at this time were negative for malaria, and blood cultures were sterile. There was a slightly positive Widal reaction. The patient's drowsiness increased, and he had the characteristic typhoid breath. The spleen was distinctly palpable and he had considerable tympanites.

A delayed complete history revealed the fact that typhoid fever had developed in several persons in the immediate vicinity of the patient's home.

The course of the typhoid was uneventful, and at the end of four weeks the patient's temperature returned to normal. Without warning, after five days with the temperature normal, marked cellulitis developed at the back of the right ear. The middle ear also started to drain, the pus being of a thick and creamy consistency. No pain was present, and the temperature was 101 F., with a leukocyte count of 11,600.

Two days later, this postauricular abscess was drained and the underlying infected mastoid process was cleaned out. The bone was soft and the cells were filled with pus. The zygoma was extensively involved, but no dural or sigmoid sinus exposure was made. Cultures from this bone showed staphylococci. The wound healed promptly and uneventfully.

COMMENT

At the first admission for tonsillectomy this boy had typhoid fever, although he had none of the clinical observations and had a normal preoperative temperature.

I do not believe that there was any relation between the early chills and high fever to the original earache. Certainly the ear infection at this time was very mild. Typhoid fever is frequently ushered in by chills, but it is rare for the chills to continue.[3] Coleman [4] states that continued chills are induced in typhoid by antipyretics. This patient was receiving 5 grains of acetyl salicylic acid every four hours.

While sinus thrombosis may be associated with a dry middle ear, leukopenia and negative mastoid observations, the association is so rare that it is worthy of careful consideration.

The characteristic typhoid breath, the drowsiness, leukopenia, palpable spleen, positive Widal reaction, and finally the history of other cases of typhoid, clinched my diagnosis that the boy had typhoid fever and not sigmoid sinus thrombosis.

The fact that the mastoid process finally became infected and required operation is not unusual. One of the common complications of typhoid fever is the late formation of abscesses. That the patient was immediately transferred to the ear service as soon as he develcped earache, chills and fever, is not surprising. In my experience, the association of high fever and chills with ear pain is always considered as proof positive of sigmoid sinus thrombosis by the pediatrician.

917 Rialto Building.

3. Holt, L. Emmet: Diseases of Infancy and Childhood, ed. 6, New York, D. Appleton & Company, 1915, p. 1011.

4. Coleman, Warren: Nelson Loose Leaf Living Medicine, Vol. I, p. 150.

ZYGOMATIC MASTOIDITIS WITHOUT CLINICAL OTITIS MEDIA*

M. L. BREITSTEIN, M.D.
BALTIMORE

That zygomatic mastoiditis is always part of a widespread mastoid infection has not been sufficiently emphasized. Mastoiditis does occur without evident otitis media, but it is very unusual to have such a case in which the first sign of mastoiditis is swelling over the zygoma. For these reasons the following case is reported:

REPORT OF CASE

J. W., a girl, aged 6, was first seen, May 26, 1924. For about one year the child had complained of a sensation of heaviness about the right ear. This discomfort occurred at irregular intervals and lasted for a day or so; there never had been any otorrhea. Up to a month prior to examination, the child had been well and active. Recently, however, her mother had noticed some languor and general lassitude. On examination, the left ear was found to be normal; the right canal was clear; there was no mastoid tenderness, and the drum was intact but of poor luster. There was no congestion or swelling. There was, however, a definite pulsation, which appeared so much like pulsating fluid that cotton was inserted. However, when it was withdrawn, the cotton was found to be dry and the pulsation continued. A detailed hearing test revealed no defect.

The mother was requested to bring the child back at the end of a week or sooner if there were any pain. However, the patient was not seen again until July 7, 1924, that is, forty-three days afterward. The mother explained that the child had been perfectly well in the interim and she saw no reason for bringing her in before, but she had noted the sudden appearance of a swelling about the right mandibular joint. The child's general condition seemed excellent; she was bright and apparently entirely unaffected by the ear condition. A circumscribed swelling about the size of a quarter could be seen just over the region of the right mandibular joint. There was no fluctuation. The swelling was exquisitely sensitive and opening the mouth was very painful. There was no swelling over the mastoid, nor was there any mastoid tenderness. The external auditory canal was of normal appearance; the drum was dull but not congested. The pulsation which had been noted at the last examination was not visible. The next day the swelling had become more marked, involving the mastoid region posteriorly and extending anteriorly to the eye. A paracentesis was performed, but no fluid was obtained. The roentgen-ray report was as follows: "The film of the left side is normal. The right mastoid shows absence of some of the mastoid cells, particularly the lower cells and those toward the tip; there is some new bone formation in place of the cells."

As the swelling became steadily worse, a mastoidectomy was performed July 11. The usual postauricular incision was made. The soft tissue was found to be greatly edematous; however, it was easily retracted and the cortex, which was soft, was exposed. The hemorrhagic appearance of the cortex was very striking, particularly in the antrum region. Immediately on its removal a pulsating hemorrhagic fluid appeared. The entire mastoid showed extensive destruction; at the tip there was sequestrum formation, but nowhere in the mastoid process was there

* Read before the Annual Meeting of the Medical and Chirurgical Faculty of Maryland, 1925.

frank pus. The antrum was also free of pus. In the temporal region the dura was found to be exposed for an area of about 2 sq. cm. There were no granulations on the dura. As soon as the zygomatic cells were opened there was a gush of about 2 drachms of thick pus. Bacteriologic culture showed *Streptococcus hemolyticus*. Drains of gauze and rubber tissue were led from the zygomatic region and from the antrum respectively. A narrow drain of plain gauze was inserted in the canal. The convalescence was uneventful, and Aug. 30, 1924, the patient was discharged entirely well.

LITERATURE

In 1920, Seligmann [1] reported four cases of zygomatic swelling in acute mastoiditis, and to this number he added the report of three other cases in the German literature. Seligmann estimates zygomatic swelling as occurring in 4 per cent of the clinic cases of acute mastoiditis. I believe this is too high an estimate. Of the cases reported, three occurred in children under 10 years of age and one in a woman of 20. Rankin [2] reported three cases of zygomatic mastoiditis, one of which occurred in a woman aged 32, and two in children under 2 years of age. It is interesting to note that the great majority of these cases were in females below 10 years of age, and that in all of them extensive involvement of the mastoid was found at operation. Hempstead [3] reported three cases of mastoiditis without apparent involvement of the middle ear, from the Mayo Clinic in 1923. In the same period of observation, there were 500 cases of mastoiditis with middle ear involvement. Hempstead was able to find reports of forty-one cases of mastoiditis without apparent middle ear involvement in the English and American literature. In addition to the cases reported by Hempstead, I have found reports of six other cases in which there was a mastoiditis without concomitant middle ear involvement.[4] As in zygomatic mastoiditis, the great majority of these cases occurred in children.

COMMENT

Suppurative inflammation of the pneumatic spaces in the temporal bone is subdivided on an arbitrary basis in accordance with the focal manifestation present. As a matter of fact, practically all middle ear infections involve the mastoid to some degree. There are exceptions, to be sure. Cases have been observed in which the middle ear has been

1. Seligmann, A.: Med. Klin. **16**:471 (May 2) 1920.

2. Rankin, Nicol: J. Laryng. & Otol. **38**:192 (April) 1923.

3. Hempstead, B. E.: Mastoiditis Without Apparent Involvement of the Middle Ear, J. A. M. A. **81**:1266 (Oct. 13) 1923.

4. Salinger, S.: Laryngoscope **30**:573 (Sept.) 1920. Katin, Tartzev: Case of Primary Mastoiditis Complicated by Meningitis, Wratschebnaja Gaza. no. 13, 1923, abstr. Zentralbl. f. Hals-, Nasen- u. Ohrenh. **111**, 1922. Chavanne, F.: Deux cas de mastoidite primitive, Proceedings Tenth International Otological Congress, Paris **6**:575, 1922, quoted from Zentralbl. f. Hals-, Nasen- u. Ohrenh. **111**:426, 1922. Heggie, N. M., and Knauer, W. J.: Mastoiditis Without Apparent Involvement of the Middle Ear, J. A. M. A. **82**:1044 (March 29) 1924.

so virulently infected as to cause meningitis and death without any mastoid involvement, as determined clinically or even at necropsy (Trivas [5]). In the case just reported, there was an extensive mastoiditis with no clinical evidence of existing otitis media. It would seem more rational to regard inflammation in the pneumatic spaces in the temporal bone as one disease regardless of the focal symptoms. It is conceivable that the mastoid can be infected primarily by way of the blood stream as in tuberculosis and osteomyelitis. Practically, however, this rarely, if ever, occurs. As the great majority of the cases of so-called primary mastoiditis occur in children, it is probable that the drainage from the middle ear through the short wide eustachian tube is good enough to prevent an accumulation of pus. In the case I report, I believe otitis media was present at the onset. Simultaneously with middle ear inflammation, a similar inflammation of the mastoid mucous membrane was also present. It is conceivable that this mastoid infection became walled off from the middle ear by a swelling of the mucous membrane lining the connection with the antrum. The otitis media apparently healed spontaneously, leaving the walled off mastoid infection. I do not believe this is a case of primary mastoiditis, but rather a case of mastoiditis without evidence of a concomitant otitis media. Investigation of the microscopic pathologic changes of the middle ear and drum indicates that infection of the middle ear and drum occurs in two ways. In the first place, there is the well recognized extension of pus up through the eustachian tube. Microscopic sections of such cases show the middle ear filled with pus and the drum membrane undergoing acute inflammatory changes. In microscopic sections of other cases there is no pus in the middle ear, but the drum and the mucous membrane of the middle ear are acutely inflamed. This type of infection is undoubtedly carried by the lymphatics.

The zygomatic cells are more numerous in childhood. This explains the greater frequency of zygomatic mastoiditis in children. It is reasonable to assume that in most cases of chronic mastoiditis there would be some involvement of zygomatic cells. The cause of unsatisfactory healing of mastoids on which operation has been performed is often the neglect of these particular cells.

Diagnosis.—The characteristic picture of a zygomatic swelling, together with an acute otitis media and mastoid tenderness, is, of course, clear cut and should give no difficulty in diagnosis. On the other hand, a zygomatic swelling without evidence of middle ear or mastoid involvement offers a nice differential diagnostic problem. Conditions to be considered are: 1. Tuberculous bone disease. 2. Syphilitic bone disease. 3. Gumma. 4. Parotitis. 5. Infection of the pre-auricular

5. Trivas: Rev. de laryngol. **44**:153, 1923.

lymph glands. As a diagnostic aid it is important to remember that although the swelling and tenderness may be limited to the zygoma in the early stages of zygomatic mastoiditis, usually the tenderness and the swelling eventually extend over the mastoid area.

The roentgen ray can be of great help. The otologist should not rely solely on the roentgen ray for the diagnosis, but should regard it simply as an aid in the study of the case. It is important for the otologist to interpret the roentgenogram himself—and in the light of his physical observations.

Treatment.—According to Luc [6] and others,[7] these unusual abscesses are to be regarded as apart from a generalized mastoiditis. Luc has assumed that bacteria, particularly pneumococcus, pass from the pharynx through the middle ear, without causing suppurative otitis media, and lodge under the periosteum. Here they cause a circumscribed periostitis. Accordingly, he would treat this condition by making a sort of Wilde incision. Such a procedure must prove ineffectual. The condition is essentially a mastoiditis, and the only rational treatment is mastoidectomy with particular attention to the zygomatic cells.

It is well to emphasize two important anatomic relations in a consideration of the surgery of the zygomatic region. The middle fossa of the skull is deepest at this point, so that the brain is in close proximity. A too vigorous pursuit of zygomatic cells can lead into the maxillary glenoid fossa, with a possible ankylosis.

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6. Katz-Blumenfeld: Handb. d. spez. Chir. d. Ohres u. d. Oberen. Lŭft. **2**:348.

7. Ann. d. mal. de l'oreille, du larynx **36,** 1910.

PHENOBARBITAL IN THE PROPHYLAXIS AND TREATMENT OF ACUTE COCAINE INTOXICATIONS

A PRELIMINARY REPORT OF ITS CLINICAL TRIAL *

M. REESE GUTTMAN, M.D.
CHICAGO

The use of cocaine in rhinologic practice is so generalized and of such frequent occurrence as to present great potentialities for overdosage and intoxications. It is a matter of common experience for one doing extensive intranasal surgery to be confronted by a so-called cocaine "reaction," and this is especially true when using "flake" anesthesia. Often people are encountered who exhibit an idiosyncrasy to the drug, and who, consequently, are susceptible even to minute amounts.

The intoxication is manifested usually by loquacity, restlessness, tremor and cardiac palpitation. There is also present a peculiar writhing of the tongue and the face. This hyperactivity of the lingual and facial musculature may become so troublesome as to interfere seriously with the necessary intranasal manipulations. Not rarely, the symptoms of mild intoxication noted in the foregoing may progress to those associated with profound depression, combined with cold clammy sweat, pallor and even syncope which may terminate in exitus.

Of late, the Council on Pharmacy and Chemistry of the American Medical Association has issued a statement decrying the promiscuous use of cocaine, especially in the form of "flakes" and in greater concentrations than 20 per cent. But while measures such as these may reduce the number of intoxications resulting from overdosage by a careless operator, they will not effect to any material extent the ill results that follow its use in hypersusceptible patients.

At the clinic, we have continued the use of "flakes," but the associate physicians are carefully trained in this application. Only a definitely measured quantity (1 grain) and no more is used, no matter how poor the subsequent anesthesia may be. Consequently, most of the "reactions" we have experienced are those due to hypersusceptibility in the patient, and for the most part are of the milder type.

Recently, Tatum, Atkinson and Collins [1] have reported on the experimental use of the barbituric acid hypnotics as they affected the minimal lethal doze of cocaine in various animals used in laboratory experiments.

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* From the clinic of Drs. Joseph C. Beck and Harry L. Pollack.

1. Tatum, A. L.; Atkinson, A. J., and Collins, K. H.: J. Pharm. & Exper. Therap. **26**:325 (Dec.) 1925.

They used a mixture of sodium barbital and a saturated solution of paraldehyde. They found that a prophylactic administration of the mixture raised the tolerance several fold to subcutaneous injections of cocaine. They also show that the convulsions and death that occur from cocaine poisoning result mainly from overstimulation of the cerebral cortex. Phenobarbital, one of the most popular barbituric hypnotics, is well known as a cerebral depressant.

These facts led us to use the drug in the following cases, one of which was a case of cocaine sensitiveness.

REPORT OF CASES

CASE 1.—B. S. a woman, aged 24, complained of typical "lower half headache," with pain radiating along the zygoma, behind the ear and down the neck. A diagnosis of Sluder's syndrome was made, and, in order to substantiate it, a topical application of 20 per cent solution of cocaine was made to the region of the nasal ganglion on the affected side. In a few moments the patient became decidedly ill, and marked pallor together with cold sweat appeared. She complained of dyspnea and said that "her heart was running away." A tablet of 1½ grains (0.1 Gm.) of phenobarbital was administered, with complete relief in a few minutes. She was allowed to go home and returned one week later. This time, after swallowing a tablet of phenobarbital, no untoward results appeared after topical application of cocaine fifteen minutes later.

Since that time, we have had numerous occasions to observe the effect of phenobarbital on "reactions" of varying degrees, and have noticed complete cessation of symptoms within five to ten minutes after exhibition of the drug. This seeming success led us to the use of phenobarbital in the rôle of a prophylactic agent. As a matter of routine in the past, we have given a preoperative hypodermic of morphine and atropine. In a series of cases, we have substituted 3 grains (0.2 Gm.) of phenobarbital by mouth thirty minutes before operation instead. Although the number of cases in this series is small, we have been impressed by the lack of "reactions" and by having more manageable, although slightly drowsy, patients. It has been especially gratifying to observe that the troublesome lingual and facial writhing has disappeared. Although we have had no serious results from the use of cocaine "flakes," we would not hesitate to use phenobarbital intravenously if ever confronted by a serious intoxication or collapse. A detailed statistical and comparative study will be reported in full later. A few illustrative case reports are appended.

CASE 2.—B. K., a man, aged 21, was seen March 3, 1926, complaining of difficulty in nasal respiration. Examination showed an extremely deflected septum and enlarged inferior turbinates. Submucous resection was advised. One week later the patient appeared for operation. A few moments after application of the "flakes" he became extremely loquacious, could not keep still, began to whistle and was unable to keep his tongue quiet. One tablet of 1½ grains (0.1 Gm.) of phenobarbital caused cessation of symptoms in about five minutes. One month later the patient returned, claiming that he experienced no relief from the pro-

cedure, and was advised to have the enlarged turbinates crushed. Thirty minutes before operation he was given 3 grains (0.2 Gm.) of phenobarbital by mouth, and the use of cocaine "flakes" caused no symptoms of intoxications.

In one case, the condition was thought to be a toxic reaction to cocaine which did not respond to phenobarbital; later it proved to be a manifestation of hysteria.

CASE 3.—C. J., a woman, entered complaining of nasal discharge. The roentgenogram disclosed a clouded antrum and ethmoids, and the patient was advised to have an intranasal procedure. A few moments after application of the anesthetic, she became tremulous, began to breathe deeply and complained of a choking sensation about the throat. Phenobarbital in the usual dose produced no effect, although repeated twice at twenty minute intervals. Dr. Beck was of the opinion that she was purely hysterical, and this was subsequently confirmed by a neurologist, Dr. Neymann, who, on examining her, found absent corneal and pharyngeal reflexes and made a diagnosis of hysteria.

CASE 4.—T. S., a man, aged 23, a medical student, complained of nasal discharge. Examination showed pus in the nose and a deflected septum. The right antrum was clouded, as seen by roentgenogram and by transillumination. He was advised to have the antrum irrigated. The application of pledgets of cotton saturated with 20 per cent cocaine caused a severe reaction, with pallor, sweat and tachycardia. These symptoms responded readily to the use of 1½ grains (0.1 Gm.) of phenobarbital. He was advised later to have a submucous resection. Thirty minutes previous to operation, he was given 3 grains of phenobarbital and 20 per cent cocaine was cautiously tried, with no bad results. Cocaine "flakes" then applied caused no unusual symptoms.

CONCLUSIONS

1. Phenobarbital is a very valuable drug in the treatment of acute cocaine intoxications.

2. It may be used prophylactically, before cocaine is used for local anesthesia, in subjects known to be sensitive to the latter.

3. It may supplant morphine as a preoperative sedative, and thus may act also in a prophylactic capacity.

4. Its intravenous use, as suggested by Tatum, Atkinson and Collins, in cases of severe intoxications or collapse due to cocaine is indicated on a rational basis.

2551 N. Clark Street.

AN EASIER METHOD OF INTRODUCING THE ESOPHAGOSCOPE *

M. JOSEPH MANDELBAUM, M.D.
NEW YORK

In performing an esophagoscopy, nothing can be so disconcerting as a persistently contracted cricopharyngeus muscle. Not only the novice, but occasionally even the more experienced operator, meets cases in which, despite preliminary morphinization and properly applied cocainization, the tightly constricted entrance to the esophagus (due to a highly developed or unduly contracted cricopharyngeus muscle), if not impossible to pass, is provocative of great distress to the patient and embarrassment to the esophagoscopist.

Cases are encountered, fortunately seldom, in which general narcosis is necessary to produce sufficient relaxation of the cricopharyngeus before the esophagoscope can be introduced.

As particularly emphasized by Chevalier Jackson, undue force in introducing the esophagoscope is dangerous; serious damage to the tissues, and even fatal perforation may ensue. The most difficult phase of the procedure is the passage of the tube past the cricopharyngeus muscle. As Jackson also properly advocates, blind bouginage or passage of the esophagoscope or other instruments into the esophagus without a full view of the structures beyond the distally lighted opening of the tube is an unnecessary and dangerous procedure.

EXPERIMENTATION

A number of cases with unusually resistant cricopharyngeus spasm led to the trial of a method to introduce the esophagoscope, analogous to the passage of a styletted urethral sound. This method meets every need of scientific esophagoscopy.

As yet, cases have not been seen in which proper indication for esophagoscopy existed when it was not possible to pass the tube by this technic.

PREPARATION AND ARMAMENTARIUM

The patient is prepared in the usual manner: History, physical examination, roentgen-ray examination, preliminary preparation, morphinization by hypodermic when indicated, and topical application of cocaine to the pyriform sinuses. The patient assumes the usual posture with head held in proper position. The usual instruments are at hand. The only extra armamentarium needed is one or two small sized (8 or

* From the Bronchoscopic Clinic, Hospital for Joint Diseases.

10, French scale) esophageal bougies. The Jackson type with flexible silk woven tip terminating in a slightly-rounded, bulbous point is employed.

TECHNIC

The esophagoscope is introduced in the usual manner, and passed into the left pyriform sinus down "to" and "gently" against the upper margin of the constricted cricopharyngeus muscle.

The nurse or assistant is at hand with the esophageal bougie. Under full vision, as soon as the slightest resistance to further passage of the esophagoscope is felt, the esophagoscope is held in the left hand and is kept gently fixed in this location, just at the constricted lumen. The bougie, the upper end of which is held between the thumb and forefinger of the right hand, is rapidly passed down the tube "to" the constricted ring of the cricopharyngeus muscle. As soon as the bougie meets with this point of resistance, it is "gently" passed about 0.5 cm. beyond the distal opening of the esophagoscope, and then gently "withdrawn" about the same distance.

By gently withdrawing the bougie a very short distance, usually about 0.5 cm., and again passing it gently downward about 0.5 cm. to 0.7 cm. against the contracted muscle, the latter will almost invariably become momentarily relaxed, and the upper circular fibers of the cricopharyngeus will grasp the point of the bougie. It may be necessary to repeat this once or twice before the cricopharyngeus relaxes. One should then wait a moment, when the muscle will relax and permit the gentle passage of the bougie another 0.5 cm.; as this occurs, the esophagoscope with the "gentlest" pressure will "ride" over the preceding tip of the bougie, past the cricopharyngeus into the esophagus. The bougie is then withdrawn and the examination continued as usual. Absolutely no force is required and the procedure is completed with practically no gagging, choking or other discomfort to the patient.

The operator frequently is gratified, after esophagoscopy by this method, by the voluntary expressions of patients on whom esophagoscopy had previously been performed. They state that by this method all dread or discomfort of this most difficult phase of the procedure is eliminated.

By way of caution, it is advised that in cases of bleeding neoplasms of the hypopharynx, and particularly in those involving the pyriform sinus, preliminary astringent swabbing of the ulcerated or bleeding surfaces, secretions from which might prevent complete visualization. Such areas should be gently swabbed with full strength epinephrine-chloride solution, or one-half strength liquid thromboplastin via the esophagoscope before the bougie is passed.

CONTRAINDICATIONS

In cases of esophageal diverticula high up in the region of the cricopharyngeus, as well as of high or low obstruction of the espohagus due to extraneous pressure such as produced by enlarged thyroid, mediastinal growths, and other agents, extra caution should be taken in advancing the esophagoscope along the bougie.

As in all other types of "nonpassable" strictures of the esophagus, at whatever level, this method as well as the orthodox procedure of esophagoscopy is contraindicated.

In conclusion, it is considered safe to recommend this method of esophagoscopy as an easier and more satisfactory procedure to both the patient and the esophagoscopist. Undoubtedly this method has been previously utilized by others in this field, but as it was encountered in this instance without previous knowledge, and as no mention of it was found in the literature, there seemed to be justification in passing it on as a pleasing innovation to the technic of esophagoscopy.

125 W. Seventy-Second Street.

PATHOLOGY OF SPHENO-ETHMOIDAL SINUSITIS*

BERNARD J. McMAHON, M.D., M.Sc. (Med.)
ST. LOUIS

In the treatment of spheno-ethmoidal sinusitis, whether conservative or radical, we are only too frequently at a loss to explain, not only to the patient but to ourselves as well, the vagaries which we obtain in our results. It is only when we stop to think of the many types of tissues making up the structures of the middle and superior turbinates and the walls of the posterior sinuses, and of the multiplicity of pathologic changes which may take place within this small area, that we begin to appreciate why our results are so confusing.

We ask ourselves, are the referred pains caused by pressure within the sinuses by retained secretions, by pressure on the nerve by edema or scar tissue, by an inflammation of the nerves themselves, by simple inflammation of the mucosa, or by the bone changes? Is optic neuritis the result of similar changes? Has the age or the sex of the patient any bearing on his or her malady, and on the result of treatment according to the microscopic picture?

It is, consequently, my intention in this survey of seventy cases, extending over a period of eighteen months, to approach these questions from a pathologist's point of view, in order to determine whether or not there is any relationship between the symptoms and the microscopic pathology, and the microscopic pathology and the end-results of operations on these sinuses.

PREPARATION OF TISSUE

The middle turbinate and cell walls were placed in 10 per cent solution of formaldehyde immediately after operation and fixed for twenty-four hours. The turbinate was sectioned in two planes horizontally and sagittally, and with the cell walls was placed in a decalcifying solution composed of 150 cc. of absolute alcohol, 150 cc. of tap water, 15 cc. of concentrated hydrochloric acid, and left in this solution until the bone was soft. These were then washed in tap water for twenty-four hours, dehydrated routinely, imbedded in paraffin, cut and stained with hemotoxylin (Harris') and eosin.

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* Candidate's thesis for the degree of Master of Medical Science in Otolaryngology, Graduate School of Medicine, University of Pennsylvania, Philadelphia, conferred June, 1926.

* Executed under the extramural preceptorship of Dr. Greenfield Sluder professor of Otolaryngology, Washington University School of Medicine, St. Louis.

NORMAL HISTOLOGY OF TURBINATES AND POSTERIOR SINUSES

Before taking up the pathologic histology of the nose, a brief presentation of the normal histology may serve to refresh our minds and establish a more definite contrast. Because of the inability to procure adequate normal specimens, I have selected five illustrations from Bauer and Beck's Atlas.[1]

PATHOLOGIC HISTOLOGY

Epithelium.—The epithelium of the turbinate, which is normally stratified and columnar in type, undergoes thickening by one or all of three ways, namely, the multiplication of the layers of columnar or squamous cells, the distention of goblet cells or the metaplasia of the columnar into stratified squamous epithelium. This flattening may be limited to the two or three more superficial layers, or it may involve all of the layers down to the basement membrane.

Metaplasia seems to occur more often at the anterior end and at points of convexity of the turbinate on the medial aspect, leading us to believe that it is in response to direct irritation caused by dust particles, smoke and gases carried by the inspired air. On the other hand, the epithelium may be undergoing disintegration, which is characterized by a ragged appearing line of cells or single row of nuclei on a thickened basement membrane. This process of disintegration is frequently accompanied by an infiltration of lymphocytes and micro-organisms, but more often it is the result of friction caused by the contact of the turbinate with the septum. There may also be a deposit of a nonstaining layer of mucus on the cells, occurring more frequently along the posterior half and the lateral aspect of the turbinate in regions which have been subjected to the constant moisture of irritating secretions flowing over them from the upper sinuses. While it is difficult to associate any of these changes with a specific clinical condition, as they may, and usually do, coexist in any long-standing intranasal infection of the posterior sinuses, the mucoid changes seem to be most apparent in the patients with asthma or in those prone to frequent acute excerbations of a low-grade chronic infectious process. While the epithelium of the ethmoidal cells and sphenoidal sinus has undergone moderate thickening in most cases, this is due in the majority to the superficial polypoid changes rather than to a direct multiplication of cell layers. In very few cases were the changes in this epithelium as profound as in that of the turbinates. Metaplasia was not observed in the mucosa of the sinuses.

1. Bauer and Beck: Atlas der Histopathologie der Naze und Ihrer und Nebenholen.

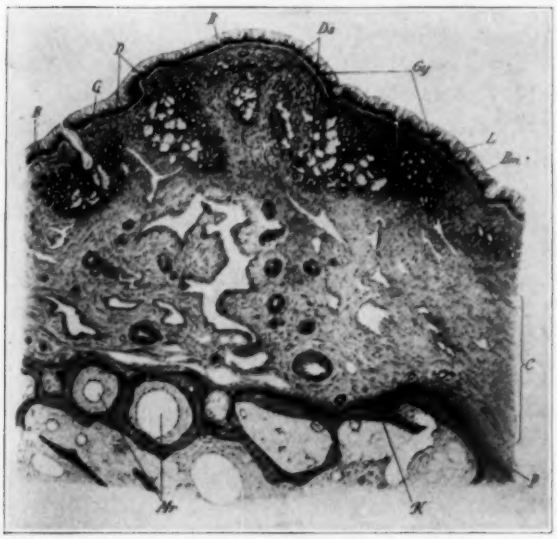

Fig. 1.—Normal middle turbinate (anterior end). *Cy*, cylindrical epithelium; *B*, goblet cells; *Bm*, basement membrane; *D*, mixed glands; *Ds*, serous glands; *G*, capillary blood vessels; *L*, round cells; *C*, vascular layer; *P*, periosteum; *K*, bone; *Mr*, medullary space.

Fig. 2.—Normal superior turbinate (posterior end). *Cy*, cylindrical epithelium; *R*, olfactory cell; *K*, bone; *Dr*, glandular layer; *P*, periosteum; *C*, cavernous bodies; *Bm*, basement membrane.

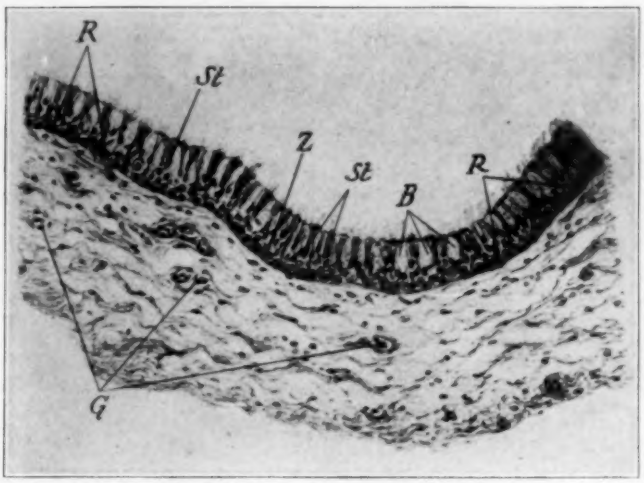

Fig. 3.—Normal ethmoidal mucosa. *Z*, high cylindrical cells; *St*, supporting cell; *R*, olfactory epithelium; *B*, goblet cells; *G*, blood vessels.

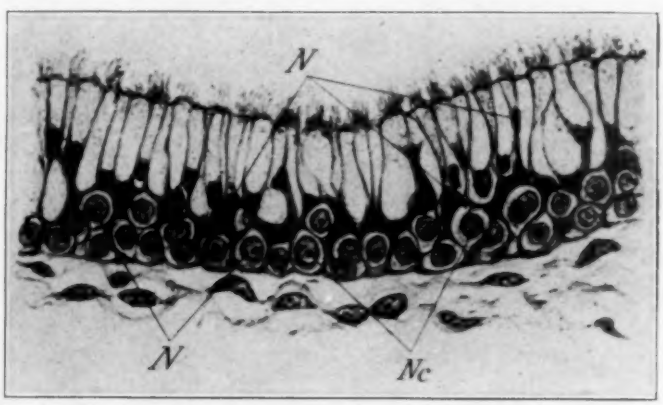

Fig. 4.—Normal olfactory region. *N*, round nuclei; *Nc*, nucleoli.

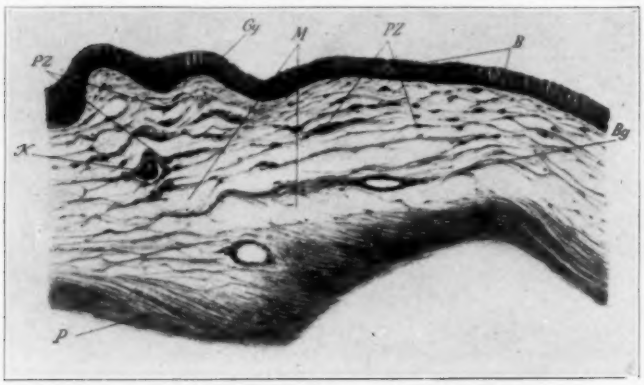

Fig. 5.—Normal mucosa of sphenoid. *Cy*, cylindrical epithelium; *B*, goblet cells; *Bg*, connective tissue; *M*, reticular tissue; *P*, periosteum; *PZ*, pigment cells; *K*, lime salt deposit.

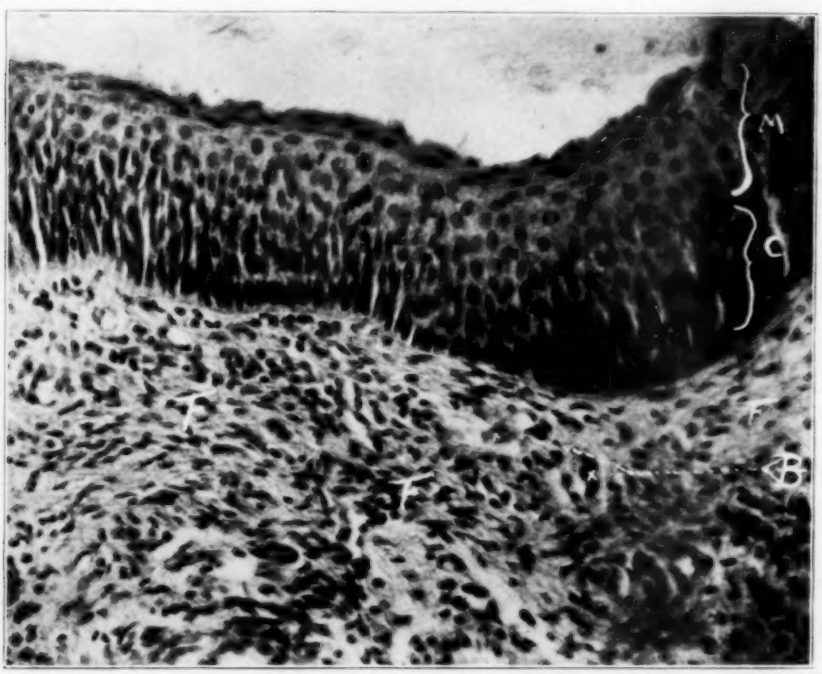

Fig. 6 (case 39).—Metaplasia of epithelium of turbinate, with extreme fibrosis of tunica propria. *M*, metaplastic cells; *C*, columnar cells; *F*, fibrous tissue; *B*, blood capillaries.

BASEMENT MEMBRANE

This structure could invariably be differentiated in the turbinates, being found extremely thickened in some specimens, and moderately so in most of them. It usually retained its homogeneous pale pink appearance, and was frequently infiltrated by bacteria or lymphocytes. The thickening was most marked when the epithelium was sloughed, the membrane assuming a fibrous-like aspect. The basement membrane was not always demonstrable in the mucosa of the ethmoidal cells or sphenoid, though in a few cases it was definitely thickened.

TUNICA PROPRIA

The three outstanding features of this stratum of the mucosa were the edema, the fibrosis and the round cell infiltration. The edema was more pronounced in the superficial portions in both the turbinates and the cell walls, and usually most pronounced at the anterior extremity of the former, though it frequently involved the entire tunica down to the periosteum being invaded by strands of connective tissue in the deeper portions. In a great many specimens, the connective tissue was more pronounced in the areas adjacent to the bone, extending outward until the tunica consisted almost entirely of dense fibrous tissue (fig. 6). The round cells, including lymphocytes, plasma cells, young connective tissue cells, and at times large numbers of eosinophils, while invading the entire tunica, always concentrated around the glands. This infiltration varied in intensity (fig. 8 *RC, D*). The mucous glands were either dilated, this dilatation at times being sufficiently extreme to cause a rupture of the walls and a confluence of the glands, or compressed by the edema or the fibrosis. The individual gland cells of the acini and tubules likewise showed certain changes, being compressed by the intra-acinic or the intratubular pressure, or dilated by intracellular pressure, thus obliterating the lumen entirely. All of these processes resulted in a disintegration of the gland and tubule. To compensate for this, numerous small new glands seemed to be developing in other areas and appeared to be surviving or perishing according to the amount of pressure which they were able to withstand. The blood vessel walls were thickened, some very little, others a great deal, the thickening in some cases being so extreme that the lumen was almost entirely obliterated (fig. 10 *VW*). In some instances the vessel wall was apparently undergoing a vacuolar degeneration.

PERIOSTEUM

This structure varied from the normal to a very extreme thickness (fig. 8 *P*), and usually in direct ratio to the degree of adjacent fibrosis of the tunica. At times, it appeared actively inflamed and infiltrated with

red blood cells; again it would be invaded by lymphocytes or young connective tissue cells, migrating from the tunica to the bone (fig. 7 *P*). In a certain number of instances the periosteum was scarcely demonstrable.

BONE

Five marked changes were found in the bone: Rarefaction, necrosis, osteomalacia, fibrosis and hyperostosis. Rarefaction was designated only as such when osteoclasts were found to be actively destroying bone.

Osteoclasts were found either singly, causing isolated erosions along

Fig. 7 (case 18).—Sinus walls (high power magnification). Marked osteoclastic activity, with mild osteoblastic activity. *Oc*, osteoclasts; *MOc*, multinuclear osteoclasts; *Ob*, osteoblasts; *B*, bone; *P*, periosteum; *Bs* space from which bone has been absorbed.

the edge of the bone, or in large numbers, causing a massive destruction from all sides. The osteoclast, as such, was scarcely ever characteristic, i. e., distinguished morphologically from the young connective tissue cell or an osteoblast. Occasionally, it appeared as a large stellate, multinucleate cell (fig. 7 *MOc, Oc*), but this was the exception rather than the rule. At times, it required the most careful study to determine whether a cell was destroying bone or building it up, and in many cases we were unable to reach a final decision. Just as the action of the osteoclast was the determining factor in rarefaction, the action of the

osteoblast was the criterion of hyperostosis. The osteoblasts, when characteristic, appeared as a line of dark staining rod, or short spindle shaped cells lying end to end, parallel to the surface of the bone underneath the periosteum (fig. 8 *Ob*) or to the curve of the lacunae, the bone surface being smooth and regular and showing an excess deposit of calcium in various stages of enveloping of the osteoblasts, these being enveloped in much closer approximation than occurs in normal bone building. This we termed pathologic hyperostosis. The regular arrange-

Fig. 8 (case 47).—Turbinate. Simultaneous osteoclastic and osteoblastic activity, with fibrosis of bone. *P*, periosteum (extremely thickened); *Ob* osteoblasts; *B*, bone; *Ms*, medullary space; *Oc*, osteoclastic activity from young connective tissue; *CT*, connective tissue, following osteoclasts; *Cap*, blood capillaries; *Oed*, edematous tunica propria; *RC*, round cell infiltration about dilated glands (*D*); *D*, dilated glands.

ment of the osteoblasts contrasted with the ungoverned, irregular invasion of the osteoclasts and was one of the means of differentiation (fig. 7 *Oc, Ob*).

The osteoblasts appeared to originate from two sources, the periosteum and the nonspecific connective tissue of the tunica propria; the osteoclasts, from the mass of young connective tissue cells, being transformed as they came into contact with the bone; this metamorphosis

was only functional, and not physical (figs. 7, 8 and 9). According to Von Gaza,[2] paraplastic substances which are released by the dying or dead bone cause this transformation, since necrotic and formative bone stand in such close physical and chemical relationship. Likewise, the latter statement found to be true, though not always, since bone necrosis was present in many of the specimens without any attempt at repair by the formation of new bone or connective tissue in the immediate vicinity. The osteoblasts likewise assumed their specific function in certain cases from the young connective tissue cells of the tunica, and

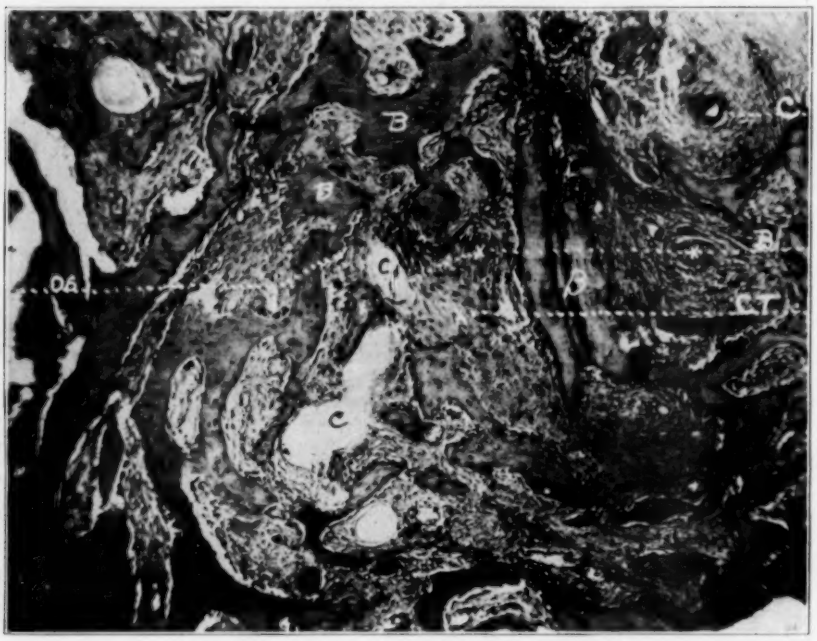

Fig. 9 (case 9).—Cell walls. Heteroplastic bone formation in young connective tissue; mild osteoblastic activity. *B*, bone; *CT*, young connective tissue cells and fibrin; *Ob*, osteoblasts; *C*, blood capillary; *BI*, bone islands.

only became differentiated as they were in the act of being enveloped by calcium (Martin[3]). The true osteoblast in most cases came from the connective tissue of the periosteum. Bancroft[4] says that the periosteum forms an adjunct, but that it is not the only form of connective

2. Gaza, von: Die Bedeutung der Gewebszerfallstoffe (Autolysate) für das regenerative Geschehen, Arch. f. klin. Chir. **121**:378, 1922.

3. Martin, B.: Zur Knochenregeneration aus dem Periost. Zur Entwicklung des Ligamentum interosseum am Unterarm und Unterschenkel, Arch. f. klin. Chir. **120**:744, 1922.

4. Bancroft, Frederic W.: Bone Repair Following Injury and Infection, Arch. Surg. **5**:646 (Nov.) 1922.

tissue capable of bone repair. I do not agree with Rohde [5] in his denial of the metaplasia of the connective tissue, either of the periosteum or the endosteum, or of nonspecific connective tissue cells of the neighborhood into osteoblasts (fig. 9). In the marrow spaces, young connective tissue cells were seen to be definitely assuming the function of osteoblasts and osteoclasts (fig. 8 *Ms*). I am inclined to agree with Louis Christophe [6] that the chemical process in the blood serum is mainly the basis of new bone formation. This is in accord with our hypothesis

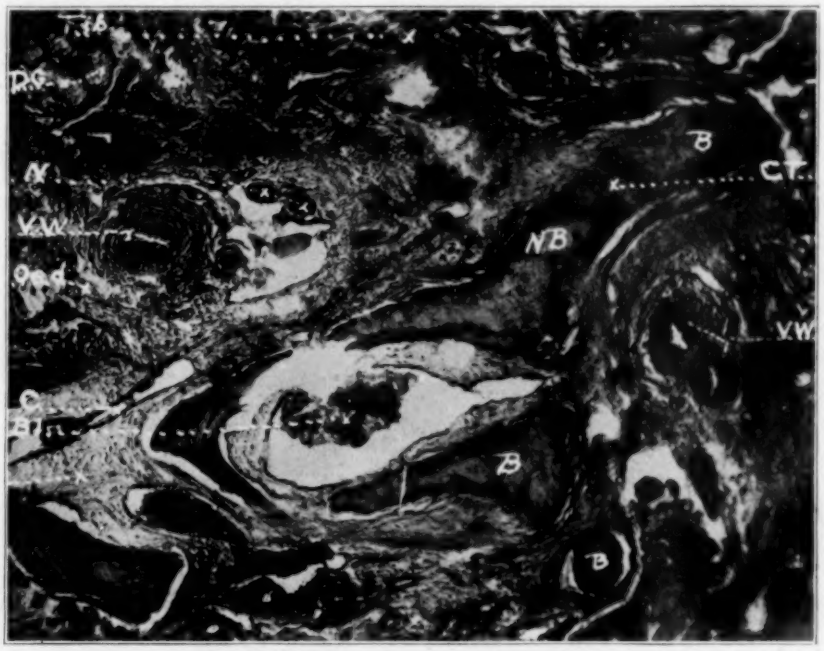

Fig. 10 (case 46).—Atrophic turbinate (low power magnification). Typical illustration of advanved generalized hyperplasia of turbinate. *B*, bone; *NB*, necrotic bone; *Ct*, connective tissue invading bone; *VW*, blood vessel walls (extremely thickened); *DG*, dilated glands; *N*, nerves; *Oed*, edematous tunica propria; *C*, blood capillary (cut lengthwise); *Bl*, free blood in blood space; *Fib*, fibrosis.

that the depression of the blood supply, following thickening of the vessel walls, leads to rarefaction and bone absorption, and ultimately to fibrosis (fig. 10).

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5. Rohde, Carl: Ueber den Ablauf der Regenerations—vorgänge am Röhrenknochen beierhaltener und geschadigter Defässversorgung, zugleich ein Beitrag uber Herkunft und Entstehungsbedingungen des Bindergewebes nach Knochenverletzungen, Arch. f. klin. Chir. **123**:530, 1923.

6. Christophe, Louis: Recherches sur les greffes d'os fixe a l'alcohol et sur le mecanisme de l'osteogenese, Arch. franco-belges de chir. **22**:257, 1923.

Necrosis was considered such when the bone took an irregular stain, having a rough, atypical appearance, with absent nuclei. It is distinguished from rarefaction in that it is usually an acellular mass action occurring centrally, while rarefaction occurs peripherally and is cellular. Osteomalacia or young connective tissue cell invasion of spontaneously decalcifying bone is a process separate and distinct from either rarefaction or necrosis, being more akin to fibrosis in its earliest stages. It presents a picture of large masses of young connective tissue cells invading poorly staining or nonstaining bone, in which the ghosts of the

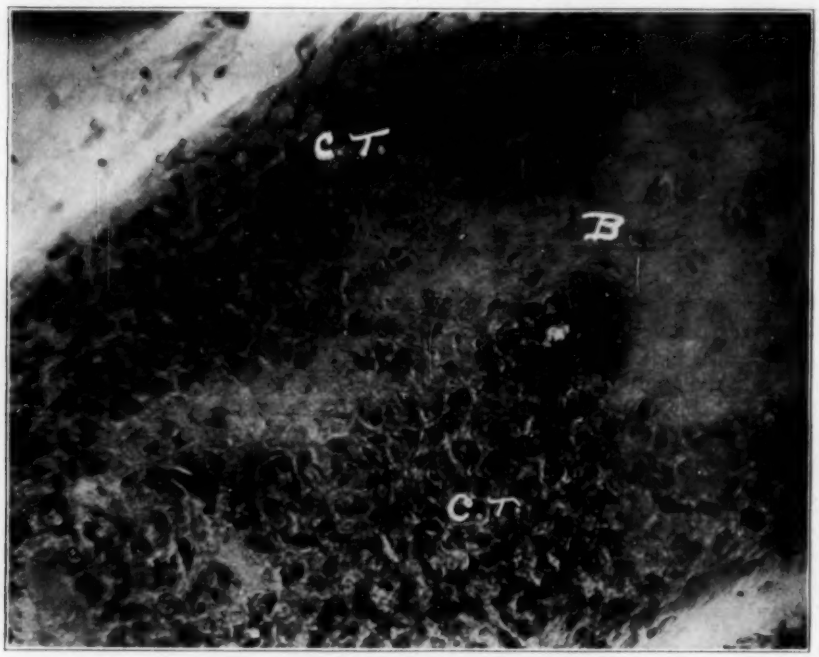

Fig. 11 (case 9).—Cell walls (high power magnification). Young connective tissue cell invasion of normal bone; osteomalacia. *B*, bone; *CT*, young connective tissue cells.

bone cells may still be seen. The connective tissue cells have apparently swarmed through the periosteum and into the decalcifying bone and completed the resorption of the calcium. Osteomalacia distinguished from necrosis shows, in the former, the calcium disappearing first and bone cells last, while in the latter the cells disappear first and the calcium last, and in rarefaction the process is much slower, the bone remaining normal until the osteoclast destroys it.

Fibrosis nearly always closely followed rarefaction, so that the erosions were quickly filled with connective tissue which extended in from

the periosteum or from the connective tissue of the tunica (fig. 8 *CT*). In many specimens, bridges of connective tissue completely separated particles of bone, or islands of bone were found completely surrounded by connective tissue (fig. 10 *CT*). These may have been instances of heteroplastic bone formation, that is, the formation of new bone separate from the old bone and periosteum, though in the same structure (figs. 9, *B* and 10 *B*).[7]

HISTOPATHOLOGY

Metaplasia of Epithelium.—I feel that this change was due entirely to extrinsic irritation rather than to any circulatory or chemical phenomena within the underlying tissue, because of the location of the metaplasia. It was always found at points at which the epithelium would be most susceptible to inhaled irritants, and did not occur over a large expanse of turbinate generally. The fact that it was never encountered in the sheltered epithelium of the ethmoidal cells further substantiates this hypothesis. It is apparently protective in nature.

Basement Membrane.—The thickening of this subepithelial layer in so many specimens, and especially when the epithelium was sloughed, leads us to ascribe to it a secondary protective function for the more delicate structure of the tunica against both mechanical irritation and invasion of bacteria.

Round Cell Infiltration About Glands.—The picture of the lymphocytic reaction around the mucous glands, leads to the belief that these were the sites of the greatest chronic inflammation and the greatest activity in combating infection (fig. 8, *RC*). This fact was borne out by the increase in secretion of groups of glands when other groups had undergone pressure atrophy. The extreme dilatation of some glands could also be ascribed to a backing up of secretion, due to their inability to drain sufficiently rapidly as a result of compression of their ducts, causing a transudation of mucus into the tunica propria, thus establishing a condition of extensive polypoid degeneration.

Thickening of the Walls of the Blood Vessels.—This thickening was one of the most constant pathologic findings, and for that reason it was considered one of the most significant (fig. 10). According to Rohde,[5] by diminishing the blood supply to a part, connective tissue formation is stimulated, especially in bone undergoing repair; consequently, reasoning "a posteriori," since connective tissue cells of all ages were so consistently found, they must have resulted to a great extent from a diminished blood supply, and the interference with the blood supply must have occurred primarily at a point at which the vessels con-

7. Gruber, George B.: Ueber heteroplastische Knochenbildung in der Muskulatur und ihrer Nachbarschaft, Bruns Beitrage z. klin. Chir. **106**:384, 1917.

TABLE 1.—*Microscopi*

Number	Epithelium				Tunica Propria							Bone					Necrosis
	Thickening	Sloughing	Polypoid Degeneration	Metaplasia	Basement Membrane, Thickening	Edema	Round Cell Infiltration	Fibrosis	Glands	Vessel Walls	Periosteum, Thickening	Osteoblastic Activity	Osteoclastic Activity	Fibrosis	Hyperostosis	Osteomalacia	
1	++	++	+	0	+++	++	+++	+++	Dil.	+++	+++	++	+	0	++	0	0
	+	+	+	0	N	+	++	+	Dil.	+++	+	+	0	0	+	0	0
2	++	0	+	0	+++	+++	+++	+++	++	+++	+++	+	++	++	0	0	0
	+	0	+	0	+	++	++	++	Com.	++	++	0	+	+	0	0	0
3	N	0	++	0	++	+	+	0	Dil.	++	++	0	+	0	0	0	0
	N	0	++	0	+	+++	++	0	Dil.	N	N	0	0	0	0	0	0
4	N	+++	0	0	+++	+++	++	+	Dil.	+++	+++	0	+	0	0	0	0
	N	+	++	0	+	++	++	0	Dil.	++	++	0	+	0	0	0	0
5	+++	+	+++	0	++	++	++	+++	Com.	++	++	0	+	+	0	0	0
	++	0	+++	0	+	++	++	+++	Com.	++	+	0	+	0	0	0	0
6	+++	0	+++	0	+++	++	++	0	D.& C.	++	++	0	+	+	0	0	0
	0	0	++	0	N	+	++	0	Dil.	++	+	0	+	0	0	0	0
7	+++	0	0	0	+++	++	+++	++	Dil.	+++	+++	0	+++	+++	0	0	0
	++	0	0	0	+	+	+++	++	Com.	+++	++	0	++	++	0	0	0
8	++	++	++	0	+++	++	+++	+++	Dil.	++	+++	+	+	0	+	0	0
	+	+	++	0	++	++	++	+++	Com.	++	++	+	+	0	+	0	0
9†	N	++	0	0	N	+++	+++	+++	Com.	++	+++	+++	0	0	+++	+++	0
10	++	+++	0	0	+++	++	+++	++	Dil.	++	0	0	+	+	0	0	0
	+	+	+	0	N	+	+++	+	Dil.	++	N	0	+	0	0	0	0
11	++	++	++	0	++	++	++	+++	Com.	+++	+++	0	++	+	0	0	0
	++	++	++	0	++	++	++	+++	Com.	+++	+++	0	+++	+	0	0	0
12	—	++	+	0	+++	+++	+ E+	+++	Com.	+++	+++	0	+++	+++	0	0	0
	—	++	+	0	+++	+++	+ E+	+++	Com.	+++	+++	0	+++	+++	0	0	0
13	—	+++	++	0	+++	+++	+++	++	Dil.	+++	+++	0	+++	+	0	0	0
	—	+++	++	0	+++	+++	+++	++	Dil.	+++	+++	0	+++	+	0	0	0
14	++	++	+++	0	+++	+++	+++	+++	Dil.	+++	++	0	+	+	0	0	0
	++	++	+++	0	+	+++	+++	+++	Dil.	+++	++	0	+	+	0	0	0
15†	++	+	0	0	+	+	++	+++	Com.	+++	++	+++	0	0	+++	0	0
16†	++	0	0	0	+	++	++	0	N	++	N	N	N	N	N	N	0
17†	+++	+++	+++	0	+++	++	++	+++	Dil.	++	0	0	0	0	0	0	0
18†	++	0	+	0	+++	+	+++	+++	D.& C.	+++	+++	0	+++	+++	0	0	0
19	+++	+++	+++	+++	+++	+++	+++	+++	D.& C.	+++	+	+++	+	0	+++	0	0
	++	+	++	0	++	+++	+++	+++	D.& C.	+++	+	+++	+	0	+++	0	0
20	+++	+++	+++	0	++	+++	+++	0	Dil.	++	+	N	N	N	N	N	0
	++	++	+++	0	+	+++	+++	0	Dil.	++	+	N	N	N	N	N	0
21	+++	0	+++	0	++	++	+++	+	Dil.	+++	+++	0	+++	+	0	0	0
	++	0	+++	0	+	++	++	0	Dil.	++	+++	0	++	+	0	0	0
22	++	+++	0	0	+++	0	++	+++	Com.	+++	++	0	+++	+++	0	0	0
	++	+	0	0	++	0	++	+++	Com.	+++	++	N	N	N	N	N	0
23	++	0	+++	0	N	+++	++	++	Com.	++	N	0	+	0	0	0	0
	++	0	+++	0	N	+++	++	++	Com.	++	N	0	+	0	0	0	0
24†	++	++	0	0	++	++	++	+++	Com.	+++	+++	0	++	0	0	0	0
25	++	0	++	0	++	+++	++	+++	+++	++	+++	0	+	+	0	+	0
	++	0	++	0	++	+++	++	+++	+++	++	+++	0	+	+	0	+	0
26	+++	++	0	++	+++	+++	+++	+++	Com.	+++	++	0	+	0	0	0	0
	+	0	0	0	N	+	++	++	Com.	++	+	0	+	0	0	0	0
27	++	+	+	0	++	0	++	++	+++	N	++	0	++	++	0	0	0
	+	0	+	0	N	+	++	++	++	+	N	0	+	+	0	0	0
28†	++	0	++	0	N	0	+++	+	N	++	+	+++	0	0	+++	0	0
29	++	0	0	++	+++	++	+++	+++	Com.	+++	++	0	+	+	0	0	0
	++	0	+	0	++	+	++	++	Com.	++	++	+	+	0	0	0	0
30	+++	0	0	+++	+++	+	+++	+++	Dil.	++	+++	0	+++	+++	0	0	0
	++	++	+++	0	+	+++	++	++	Dil.	++	N	0	+	0	0	0	0
31	++	0	++	0	+++	+	+++	+++	Com.	++	+++	0	0	+	0	0	0
	+	0	+	0	N	+++	E++	0	D.& C.	++	N	0	+	0	0	0	0
32	—	++	0	0	+	++	++	0	D.& C.	+++	+	0	+	0	0	+	0
	—	++	0	0	+	++	++	0	D.& C.	+++	+	0	+	0	0	+	0
33†	++	0	0	0	+	0	++	+++	Com.	+	N	0	0	0	0	0	0
34	++	+	+	++	N	+++	+++	+++	Dil.	+++	+++	0	+++	+++	+	0	0
	+	0	0	0	N	0	+++	++	Dil.	+++	+++	0	+++	++	0	0	0
35	++	+	0	0	++	+++	+++	+++	D.& C.	+++	++	+++	+	+	+++	0	0
	++	+	0	0	++	+++	+++	+++	D.& C.	+++	++	+++	+	+	+++	0	0
36	+++	+++	+++	+++	+++	++	++	+++	Com.	++	++	0	0	+	0	+	0
	++	0	+++	0	+	++	++	++	Com.	++	+	0	0	+	+	+	0
37	+++	0	0	++	++	++	+++	+++	Com.	+++	++	0	+	0	0	0	0
	++	++	+	0	N	++	+++	+++	Com.	+++	++	0	0	++	0	0	+
38	++	+++	++	0	+++	++	++	+++	D.& C.	++	+++	+++	+	++	+++	0	0
	++	0	++	0	+	0	++	+++	D.& C.	++	+	0	0	+	0	++	0
39	+++	0	++	++	++	0	+++	+++	Com.	+++	+++	+++	0	++	+++	0	0
	++	0	+	+	+	0	++	++	Com.	++	++	+++	0	++	+++	0	0
40	++	+	0	0	++	+	++	+	Com.	+++	++	+++	+	+	+++	0	0
	++	+	0	0	++	+	++	+	Dil.	+++	++	+++	+	++	+++	0	0

* Upper characters, turbinates; lower characters, sinus and cell walls; Dil., dilated; Com., compressed; D.& C., dilated a
compressed; N, normal; 0, none present; +, mild; ++, moderate; +++, extreme; E, eosinophils.
† Turbinates.
‡ Sinus and cell walls.

Diagnosis	Age	Sex	Type of Operation	Time Since Operation, Months	Result
Chronic inflammation, hyperplasia, hyperostosis, early rarefaction	18	F	Spheno-ethmoidal opening, alcohol injection, bilateral	18	Great improvement in all symptoms
Chronic inflammation, extreme hyperplasia, polypoid changes; bone rarefaction and fibrosis	33	F	Spheno-ethmoidal opening, right...	17	Improvement
Chronic inflammation, moderate; very early rarefaction of bone	32	F	Spheno-ethmoidal opening, bilateral	17	Improved
Chronic inflammation; acute exacerbation; hyperplasia, hyaline degeneration	18	M	Spheno-ethmoidal opening, right...	17	Immediate complete relief
Polypoid degeneration, hyperplasia; very early rarefaction and fibrosis of bone	37	M	Spheno-ethmoidal opening, right; alchohol injection left ganglion	17	Improved
Acute inflammation, early chronic changes, mild hyperplasia; early rarefaction	22	F	Spheno-ethmoidal opening, bilateral	17	Improved
Chronic inflammation, hyperplasia; bone rarefaction and fibrosis	50	F	Spheno-ethmoidal opening, bilateral	17	Not improved
Chronic inflammation, some hyperplasia and early hyperostosis	27	F	Spheno-ethmoidal repair, bilateral..	16	Improved
Chronic inflammation, edema; osteomalacia	27	F	Spheno-ethmoidal repair, bilateral..	16	Improved
Chronic inflammation, with early rarefaction	26	M	Spheno-ethmoidal opening, bilateral	16	Unknown
Chronic inflammation, moderate bone rarefaction	7	M	Spheno-ethmoidal opening, bilateral	16	Unknown
Acute, on chronic hyperplastic inflammation; advanced rarefying osteitis, fibrosis	33	F	Spheno-ethmoidal opening, bilateral	16	Unknown
Mucoid degeneration of mucosa, with fibrosis; rarefying osteitis, with fibrosis	44	F	Spheno-ethmoidal opening, bilateral	15	Improved
Chronic inflammation, fibrosis; early bone rarefaction	29	F	Spheno-ethmoidal opening, bilateral	15	Relief, except for occasional acute exacerbation
Chronic inflammation, marked hyperplasia; formation of new bone	23	M	Spheno-ethmoidal repair, bilateral; right sphenoid reopened seven months later	15	Improved; died in coma, cause unknown
Chronic inflammation	46	F	Spheno-ethmoidal opening, bilateral	15	Marked relief; tendency of sphenoids to close
Chronic inflammation, polypoid degeneration, edema	9	M	Spheno-ethmoidal opening, right....	14	Unsatisfactory
Chronic inflammation, advanced fibrosis; advanced bone rarefaction and fibrosis	32	M	Spheno-ethmoidal opening, bilateral	14	Improved
Chronic inflammation, moderate hyperplasia; extensive new bone formation	20	F	Spheno-ethmoidal opening, bilateral	14	Much improved
Chronic hypertrophy, with acute inflammation and polypoid changes	58	M	Spheno-ethmoidal opening, bilateral	14	Hearing and general health improved
Chronic hyperplasia, pus foci; active congestion, generalized; bone rarefaction, with early fibrosis	50	F	Spheno-ethmoidal openinig, bilateral	14	Unknown
Advanced hyperplasia, marked rarefaction and fibrosis of bone	47	F	Spheno-ethmoidal opening, bilateral	14	Relief from all symptoms
Chronic inflammation, edema; very early bone rarefaction	35	F	Spheno-ethmoidal opening, bilateral	14	Improved
Advanced hyperplasia; bone rarefaction and fibrosis, moderate	38	F	Spheno-ethmoidal opening, bilateral	13	Relief, although occasional acute exacerbation
Chronic inflammation; acute inflammation; early rarefying osteitis and fibrosis	28	F	Spheno-ethmoidal opening, bilateral	13	Much improved
Chronic inflammation; early rarefying osteitis	65	M	Spheno-ethmoidal opening, bilateral	13	Died of pneumonia
Chronic inflammation; moderate rarefying osteitis and fibrosis	9	M	Spheno-ethmoidal opening, left.....	13	Improved
Chronic inflammation; marked hyperostosis	9	M	Spheno-ethmoidal repair, bilateral..	12	Improved
Chronic inflammation; very early rarefying osteitis and fibrosis	26	F	Spheno-ethmoidal opening, bilateral	12	Cured
Chronic inflammation, extreme hyperplasia; marked rarefying osteitis and fibrosis	62	M	Spheno-ethmoidal opening, bilateral	12	Unknown
Chronic inflammation; acute inflammation; very early rarefying osteitis	23	F	Spheno-ethmoidal opening, bilateral	12	Slowly improved; decrease in intervals and severity
Chronic inflammation; early rarefying osteitis	27	F	Spheno-ethmoidal opening, bilateral	12	Cured; vision above normal
Chronic inflammation and fibrosis.....	27	M	Spheno-ethmoidal opening, bilateral	12	Almost immediate complete relief
Chronic inflammation, moderate rarefying osteitis and fibrosis	28	F	Spheno-ethmoidal opening, bilateral	12	Immediate complete relief
Chronic inflammation, marked hyperostosis	9	M	Spheno-ethmoidal opening, left.....	12	Improved
Chronic inflammation; early osteomalacia	23	F	Spheno-ethmoidal opening, left.....	12	Unknown
Chronic inflammation; early rarefying osteitis and necrosis of bone	41	M	Spheno-ethmoidal opening, bilateral	12	Unknown
Chronic inflammation; marked hyperostosis	62	F	Spheno-ethmoidal opening, bilateral	11	Headache cured; increase in weight
Chronic inflammation; extensive hyperostosis and new bone formation	58	F	Spheno-ethmoidal opening, bilateral	11	Improved
Chronic inflammation, moderate fibrosis; early rarefying osteitis and fibrosis	40	F	Spheno-ethmoidal opening, bilateral	11	Immediate complete relief

Number	Epithelium					Tunica Propria						Bone					
	Thickening	Sloughing	Polypoid Degeneration	Metaplasia	Basement Membrane, Thickening	Edema	Round Cell Infiltration	Fibrosis	Glands	Vessel Walls	Periosteum, Thickening	Osteoblastic Activity	Osteoclastic Activity	Fibrosis	Hyperostosis	Osteomalacia	Necrosis
41	.....	+++	0	0	N	0	+++	+++	Com.	+++	+++	+++	0	+++	+++	0	0
	.....	+++	0	0	+++	+	+++	+++	Com.	+++	N	0	+	0	0	0	0
42	++	+	0	++	++	0	.....	+++	Com.	++	++	+	+	0	0	++	0
	++	0	+	0	N	+++	+++	+++	Com.	++	++	++	++	+	+	++	0
43	++	0	+++	0	+++	+++	+++	+++	D.& C.	++	+++	+++	+	+	+++	0	0
	++	0	++	0	++	0	++	+++	D.& C.	++	++	+	+	+	++	0	0
44	+++	0	++	0	++	++	+++	++	Dil.	++	+	+	0	0	+	0	0
	++	++	+++	0	++	++	+++	+++	Dil.	+++	+	++	0	+	++	+	0
45	.....	+++	0	0	N	+++	+++	++	Dil.	++	++	+	+	0	+	0	0
	++	++	++	0	N	++	.....	0	Dil.	++	N	0	0	+	0	0	0
46	++	+++	0	0	+++	++	+++	+++	D.& C.	+++	+++	0	++	+++	0	0	0
	++	+++	0	0	+++	++	+++	+++	D.& C.	+++	+++	0	+	0	0	0	0
47	+++	0	0	+	+++	++	+++	+++	Com.	+++	+++	+++	+++	+++	+	0	0
	+++	++	++	0	+	0	++	+++	Com.	+++	++	++	++	++	+	0	0
48	++	++	0	0	++	0	+++	+++	D.& C.	++	N	0	+++	++	0	++	++
	++	++	0	0	++	++	+++	+++	D.& C.	++	N	0	++	+	0	+	+
49†	.....	+++	0	0	++	0	+++	+++	D.& C.	+++	N	0	0	0	0	+++	++
50	+++	++	++	0	++	+++	+++	++	Dil.	++	+++	0	+	0	0	0	0
	.....	+++	0	0	+++	0	+++	++	Com.	+	+++	0	+	0	0	0	0
51	+++	+++	0	0	++	+++	.....	+	Dil.	+	++	0	+	+	0	0	0
	+++	0	++	0	++	+++	.....	++	Dil.	+	++	0	+	+	0	0	0
52	++	+++	0	0	++	++	++	++	Com.	+++	+++	+	+++	+++	0	0	0
	.....	+++	0	0	++	0	+++	+++	Com.	++	+	0	0	0	0	0	0
53	+++	0	0	0	++	+	+++	++	Com.	+	N	0	+	0	0	0	0
	+	0	0	0	+	++	++	++	D.& C.	++	N	0	+	0	0	0	0
54	+++	+++	+++	0	+++	+++	+++	+++	Com.	+++	+++	+++	+++	++	++	0	0
	++	0	0	0	+++	+++	+++	+++	Com.	+++	+	0	+	+	0	0	0
55	+++	0	+++	0	+++	0	+++	+++	D.& C.	++	+++	++	+	0	++	0	0
	+++	0	+++	0	+++	0	+++	+++	D.& C.	++	+++	++	+	0	++	0	0
56	+++	0	0	+++	++	++	+++	+++	D.& C.	+++	+++	0	+++	+++	0	+++	++
	+++	+++	0	0	+++	0	+++	+++	Com.	+++	+++	0	+++	+++	0	+++	++
57	+	++	++	0	++	+++	++	++	Com.	++	+++	0	+	0	0	0	++
	+	++	++	0	++	+++	+++	++	Com.	++	+	0	++	0	0	0	+++
58‡	+++	0	0	0	++	+++	+++	++	Com.	++	+++	0	0	0	0	0	++
59	++	+++	++	0	+++	++	+++	++	D.& C.	+++	++	+	+	+	+	0	0
	++	+++	++	0	0	+++	+++	0	D.& C.	N	++	+	+	+	+	0	0
60	+++	+++	+++	++	+++	+++	+++	+++	D.& C.	+++	+++	0	+	+++	0	++	0
	+++	+++	+++	++	++	+++	+++	+++	D.& C.	++	+++	0	0	+++	0	0	++
61	++	0	0	++	++	+	+++	+++	Com.	+++	++	+++	+++	++	0	0	0
	++	0	+	0	++	++	++	++	D.& C.	++	++	+++	+++	++	0	0	0
62	++	++	++	0	+++	+	+++	+++	Dil.	++	+++	+	++	0	0	0	+++
	++	0	+++	0	—	+	+++	+++	Com.	++	N	+	++	0	0	0	+++
63	+++	0	++	++	+++	++	+++	++	D.& C.	+	++	++	++	+	++	+++	0
	—	++	0	0	+++	+++	++	++	N	N	++	+	+	+	0	0	0
64	+++	+	0	0	++	+++	+++	+++	Com.	+++	++	0	+++	++	0	0	0
	+++	+++	+++	0	++	++	+++	++	Com.	+	++	0	+++	++	0	0	0
65	+++	0	0	+++	++	++	+++	++	D.& C.	++	++	0	0	+	++	0	0
	+	0	++	0	++	++	+++	++	D.& C.	++	+	0	0	0	+	0	0
66	++	++	+	+	++	++	++	+++	D.& C.	++	+++	+	+	+	+	0	++
	+	+	+	0	+++	+++	++	+++	D.& C.	++	+	0	0	0	0	0	+
67	+++	0	0	+++	N	++	+++	+++	Com.	+++	+++	0	0	+	++	+	+++
	++	+++	0	0	+++	+++	+++	+++	Com.	+++	+++	0	0	+	++	+	+++
68	+++	0	+++	0	++	+++	++	+++	Dil.	+++	+++	0	+++	0	0	++	0
	++	++	+	0	+++	+	+	+	N	N	N	0	0	0	0	0	0
69†	+++	0	0	+++	N	++	+++	+++	N	+++	N	Eburnated			+++	0	0
70	+++	0	++	0	++	++	+++	+++	D.& C.	+++	+++	+	+++	++	0	+++	0
	+++	0	++	0	++	++	+++	+++	D.& C.	+++	+++	+	+++	++	0	+++	0

Observations—Continued

Diagnosis	Age	Sex	Type of Operation	Time Since Operation, Months	Result
Chronic inflammation; rarefying osteitis and fibrosis of turbinate	4	M	Anterior ethmoidal opening, right..	11	Immediate complete relief
Chronic inflammation; moderate rarefying osteitis, hyperostosis; osteomalacia	40	M	Spheno-ethmoidal opening, bilateral	10	Much improved, although persistence of anterior sinus infection
Chronic inflammation; early rarefying osteitis and hyperostosis	24	M	Spheno-ethmoidal opening, bilateral	10	Dizziness relieved; recurrence of headache, relieved by local treatment
Chronic inflammation; early osteofibrosis	47	M	Spheno-ethmoidal opening, bilateral; (2) left anterior and frontal opening	10	Immediate partial improvement; (2) partial improvement
Acute purulent inflammation, with fibrosis; early rarefying osteitis and fibrosis	23	M	Spheno-ethmoidal opening, bilateral; (2) bilateral frontal four months later	8	Relief for 3 months; recurrence; (2) complete relief
Chronic inflammation, advanced fibrosis; moderate rarefying osteitis, with advanced fibrosis	55	F	Spheno-ethmoidal opening, bilateral	7	Complete relief
Chronic inflammation and fibrosis, marked rarefying osteitis; hyperostosis and fibrosis	27	F	Spheno-ethmoidal opening, bilateral	9	Improved for 3 to 4 months; still has symptoms
Chronic inflammation, extreme fibrosis; extensive bone necrosis, rarefying osteitis	32	F	Spheno-ethmoidal opening, bilateral	9	Complete relief; slight recurrence on acute infection, relief on treatment
Chronic inflammation, with fibrosis; osteomalacia	45	F	Spheno-ethmoidal opening, right....	9	Improvement marked
Chronic inflammation, advanced fibrosis, polypoid degeneration; early rarefying osteitis and fibrosis	34	F	Spheno-ethmoidal opening, bilateral	9	Moderately relieved
Chronic inflammation, mucoid degeneration submucosa; early rarefying osteitis and fibrosis	20	F	Spheno-ethmoidal opening, bilateral	8	Much relieved; mild recurrence of acute infection
Subacute, on chronic inflammation; moderately advanced fibrosis; moderate generalized rarefying osteitis, fibrosis; moderate hyperostosis	13	M	Spheno-ethmoidal opening, bilateral	7	Not improved
Chronic inflammation, with moderate fibrosis; very early rarefying osteitis	53	F	Spheno-ethmoidal opening, bilateral	9	Moderate progressive relief
Chronic inflammation, advanced fibrosis, polypoid degeneration; moderately advanced rarefying osteitis, fibrosis; moderate hyperostosis	21	F	Spheno-ethmoidal opening, bilateral	7	Progressive improvement; eventual relief
Chronic inflammation, advanced fibrosis; early rarefying osteitis and hyperostosis	39	F	Spheno-ethmoidal opening, bilateral	7	Partial relief from headache
Chronic inflammation, with fibrosis; advanced rarefying osteitis and fibrosis; moderate osteomalacia	48	F	Spheno-ethmoidal opening, bilateral; later successful eye operation	6	Immediate relief of headache; improvement in all symptoms
Chronic inflammation, very early rarefying osteitis, moderate necrosis of bone	22	M	Spheno-ethmoidal opening, bilateral	4	Asthma partially relieved, worse on acute reinfection; general health imp.
Chronic inflammation, moderate fibrosis; early bone necrosis	38	F	Turbinectomy, left middle; anterior ethmoidectomy	4	Immediate progressive improvement
Chronic inflammation, early fibrosis and hyperostosis of bone	40	M	Spheno-ethmoidal opening, bilateral	3	Improvement in all symptoms
Chronic inflammation, edema, moderate bone necrosis	47	F	Spheno-ethmoidal opening, left.....	4	Gradual complete relief
Chronic inflammation, with extreme fibrosis; hyperostosis, with rarefaction and fibrosis, extreme	35	F	Spheno-ethmoidal opening, bilateral	2	Unknown
Chronic inflammation, with extreme fibrosis; rarefaction, hyperostosis, necrosis, osteomalacia	44	F	Spheno-ethmoidal opening, bilateral	2	Temporary complete relief; exacerbation on acute infection
Chronic inflammation, with moderate fibrosis; early hyperostosis and rarefaction	40	F	Spheno-ethmoidal opening, bilateral	2	Improved
Chronic inflammation, with rarefaction and osteofibrosis, advanced	44	F	Spheno-ethmoidal opening, bilateral	2	Gradual improvement
Chronic inflammation, with extreme fibrosis; moderate hyperostosis	21	F	Spheno-ethmoidal opening; bilateral	2	Relief from headache on right, left persists; chr. pur. max. sinusitis, left
Chronic inflammation, with fibrosis and early rarefaction; acute periostitis of cell walls	62	M	Spheno-ethmoidal opening, right....	6 wk.	Improved
Chronic inflammation, extreme fibrosis; extreme bone necrosis, with moderate hyperostosis	48	F	Spheno-ethmoidal opening and intranasofrontal, bilateral	5 wk.	Headache improved; right occipital persists
Chronic inflammation, fibrosis, osteomalacia of turbinate; acute edema, mild chronic inflammation of cell walls	42	M	Spheno-ethmoidal opening, left.....	1	Partial return of vision, persistent right spheno-ethmoidal infection (operation later)
Chronic inflammation, with hyperostosis, extreme	35	F	Spheno-ethmoidal opening, bilateral	1	Improved
Chronic inflammation, with extreme rarefaction and osteomalacia	35	F	Spheno-ethmoidal opening, bilateral	1	Improved

TABLE 2.—*Clinical Observations*

Number	Symptoms	Duration	Gross Pathology	Diagnosis
1	Headache; sneezing and coryza	3 years	Polypi within all posterior cells	Hay fever; spheno-ethmoidal sinusitis, purulent, bilateral
2	Bioccipital headache	4 to 5 years	Slight pus, right spheno-ethmoidal area; pyogenic membrane, right sphenoid	Hyperplastic spheno-ethmoidal sinusitis, right
3	Asthma, worse in fall, winter, and spring; large amount of mucoid nasal discharge	Several years	Polypoid tissue, sphenoidal and posterior ethmoidal sinuses	Asthma; spheno-ethmoidal sinusitis, bilateral
4	Headache, dizziness and vomiting when turned on right side	2 days	Pus, right middle and superior passages and right sphenoid	Sphenoidal and maxillary sinusitis, chronic purulent, right
5	Nasal ganglion neuralgia, left; headaches	Several years	Polypoid degeneration, mucosa of right sphenoid	Spheno-ethmoidal sinusitis, right
6	Polyarthritis	2 months	Posterior end of left middle turbinate very red; hyperplasia of septum; dilated vessels, left spheno-ethmoidal region; excess moisture, postethmoidal cells, bilateral; no pus	Spheno-ethmoidal sinusitis, bilateral; polyarthritis
7	Frontal headaches, worse with acute infection	6 years	Pus, right postethmoidal cells	Spheno-ethmoidal sinusitis, purulent, bilateral; arthritis, chonic inf.
8	Pain in left hip, with limitation of motion	10 years	Thickened mucosa, spheno-ethmoidal sinuses	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
9	Pain in left hip	10 years	Much granulation tissue, obstructing sphenoidal sinuses	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
10	Extreme stiffness of left leg	1 year	Hyperplastic tissue, all posterior sinuses; no pus	Spheno-ethmoidal sinusitis, hyperplastic, bilateral; acute inf. arthritis, left hip
11	Acute rheumatic fever	4 months	Polypoid tissue and pus, all posterior cells	Spheno-ethmoidal sinusitis, suppurative, bilateral
12	Pain and stiffness, all joints; tenderness and swelling of joints of left hand for 2 months	12 months	Hyperplastic changes, with sticky mucoid discharge, both spheno-ethmoidal recesses	Spheno-ethmoidal sinusitis, bilateral; arthritis
13	Frequent frontal headaches; failing vision	3 years	Pus and polypi, ethmoidal cells on right	Spheno-ethmoidal sinusitis, purulent, bilateral
14	Nasal stoppage, violent sneezing, watery discharge daily; moderate bifrontal headache daily for 9 months	9 years	Marked swelling, spheno-ethmoidal recesses; bilateral posterior cervical adenopathy	Spheno-ethmoidal and maxillary sinusitis, purulent, bilateral
15	Severe headache, worse on right, intermittent; constant postnasal mucoid discharge for several years	3 years	Polypoid tissue, all posterior sinuses	Spheno-ethmoidal sinusitis, purulent, bilateral
16	Nasal obstruction, tightness over bridge of nose; anosmia; impaired hearing; transient blindness on exertion	Several years	Hyperplasia, left inferior turbinate, mucoid discharge from posterior cells on right	Sphenoidal sinusitis, bilateral
17	Abthma, intervals of 2 to 3 months; recently every 10 to 14 days	7 years	Generalized hyperplastic changes, spheno-ethmoidal cells	Spheno-ethmoidal sinusitis, right
18	Pulmonary abscess, following tonsillectomy	6 months	Purulent discharge, both spheno-ethmoidal recesses; pus and pyogenic membrane, spheno-ethmoidal and postethmoidal cells	Spheno-ethmoidal sinusitis, purulent, bilateral
19	Headaches, following fall on back of head; arthritis for several years	1 year	Marked hyperplasia, thickening, with hard bone, postethmoidal cells	Spheno-ethmoidal sinusitis, purulent, bilateral

20	Mucopurulent postnasal discharge; impaired hearing for 5 to 6 years	30 years	Pyogenic membrane, polypi, and pus, all posterior cells	Pansinusitis, purulent
21	Sinusitis; headaches; multiple arthritis; occasional dizziness	Several years	Marked redness and thickening, spheno-ethmoidal recesses	Spheno-ethmoidal sinusitis, purulent, bilateral
22	Severe daily morning headaches, soreness, posterior head and neck; marked underweight; melancholy and nervous	9 years	Redness and thickening at anterior tip of middle turbinates	Spheno-ethmoidal sinusitis, purulent, bilateral
23	Intense lower half headaches, pain referred down arms to fingers; practically blind 5 months ago, improved with ocular training	4 years	Sinuses and cells lined with thick, hyperplastic mucosa	Spheno-ethmoidal sinusitis, hyperplastic, bilateral; choroiditis
24	Severe headache, duration 7 to 9 days, every 6 to 8 weeks	8 years	Pus within right sphenoid.....	Spheno-ethmoidal sinusitis, purulent, right
25	Parietal and occipital headaches, moderately severe; some deafness	Several months	Practically no anteronasal or postnasal discharge; pus, both sphenoidal sinuses	Spheno-ethmoidal sinusitis, purulent, bilateral
26	Extreme sepsis immediately following antrum operation	1 week	Mucosa throughout sinus areas inflamed, swollen; thin mucopus from all sinuses	Pansinusitis, acute suppurative; pneumonia
27	Herpes over course of second div. fifth nerve, left, recurrent	Several years	Pus, left sphenoidal sinus.....	Sphenoidal sinusitis, chronic purulent, left; ulcer, left cornea
28	Asthma	Several years	Pyogenic membrane, posterior sinuses.....	Spheno-ethmoidal sinusitis, purulent, left
29	Severe daily headache, worse in winter; multiple arthritis; lack of concentration for several years	18 months	Pyogenic membrane and pus, speno-ethmoidal sinuses; posterior cervical adenopahthy	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
30	Arthritis; enlargement of knee joints; constant purulent postnasal discharge; several bioccipital headaches for 5 to 6 years	5 years	Pus, spheno-ethmoidal recesses; chronic hyperplastic changes, same; pyogenic membrane, sphenoids	Spheno-ethmoidal sinusitis, chronic purulent, bilateral; arthritis, chronic
31	Headaches, bilateral frontal, parietal, occipital (worse in summer); gynecologic condition for several years; pain behind right eye, with vomiting	13 years	Tenderness, Ewing's point, left; swelling of both middle turbinates	Spheno-ethmoidal sinusitis, purulent, bilateral; maxillary sinusitis, purulent, bilateral
32	Pain, left eye; failing vision; constant purulent postnasal discharge for 4 weeks; severe frontal and occipital headaches for 5 to 6 years	2 weeks	Mild inflammatory reaction in sinuses; central scotoma, left	Spheno-ethmoidal sinusitis, hyperplastic, bilateral; retrobulbar neuritis (Dr. L. Post)
33	Nasal obstruction, frequent supraorbital headaches	Several years	Polpi, all posterior sinuses.....	Spheno-ethmoidal sinusitis, purulent, bilateral; deflected septum
34	Severe left frontal headache every 2 to 3 months, lasting 3 to 4 days	1 year	Slight pus in nasopharynx, pus in left middle meatus; swelling, left middle turbinate	Spheno-ethmoidal sinusitis, purulent, bilateral
35	Asthma	Several years	Pyogenic membrane, left spheno-ethmoidal sinuses	Spheno-ethmoidal sinusitis, purulent, left
36	Severe headaches; inanition, active pulmonary tuberculosis, and constant postnasal discharge for several years	2 years	Mucosa on right water-logged, both middle meatuses closed, much pus on posterior pharyngeal wall; thickened mucosa and pus, all posterior sinuses	Spheno-ethmoidal sinusitis, purulent, bilateral
37	Considerable nasal discharge; severe bifrontal headaches and dimness of vision for 2 to 3 months	Several years	Mucosa of left sphenoid thickened.....	Spheno-ethmoidal sinusitis, purulent, bilateral; optic neuritis

TABLE 2.—*Clinical Observations—Continued*

Number	Symptoms	Duration	Gross Pathology	Diagnosis
38	Recurrent attacks of cough, dizziness, and pain in head and between scapulae (very acute past 3 weeks)	3 years	Pyogenic membrane, sphenoidal sinuses.....	Spheno-ethmoidal sinusitis, purulent, bilateral
39	Severe headaches, worse in morning; profuse postnasal purulent discharge, not relieved by treatment	Several years	Pyogenic membrane, spheno-ethmoidal sinuses	Spheno-ethmoidal sinusitis, purulent, bilateral
40	Band pain about head; fetid discharge from nose on bending head forward; apprehension, nervousness	Several months	Sphenoidal sinuses filled with dark liquid pus	Spheno-ethmoidal sinusitis, purulent, bilateral
41	Bulging right eye, blackening of lid; eye grounds negative; semiconsciousness for 8 hours	24 hours	Marked swelling and redness of nasal mucosa, slight pus in right middle meatus	Orbital abscess, right
42	Severe left frontal, temporal, and occipital headache, some relief by treatment	Several years	Pyogenic membrane and pus, sphenoidal and ethmoidal cells, bilateral; extensive bone necrosis	Spheno-ethmoidal sinusitis, purulent, bilateral
43	Difficult breathing through nose, nasal discharge; suboccipital headaches, drowsiness, only partially relieved by treatment	Several years	Nasal mucosa red, congested; mucopus, spheno-ethmoidal areas	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
44	Recurrent headache and dizziness lasting 2 to 3 weeks, associated intranasal infection (not relieved by treatment)	Several years	Pyogenic membrane and pus, both sphenoidal sinuses	Spheno-ethmoidal sinusitis, purulent, bilateral; maxillary sinusitis, purulent, bilateral
45	Asthma, summer and winter type (despite several nasal operations); sensitization tests negative	Several years	Pus and granulations in sphenoid; lining removed in toto	Pansinusitis, chronic purulent, bilateral
46	Severe generalized headaches.....	Several years	Polypoid membrane, posterior sinuses.....	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
47	Cardiac disease, edema of feet and ankles.....	Several years	Marked hyperplasia and redness, with pus, both spheno-ethmoidal areas; thickened mucosa of posterior sinuses	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
48	Attacks of violent, uncontrollable sneezing, becoming progressively worse; occasional right lower half headache	1½ years	Nasal mucosa boggy and pale; some hyperplasia, spheno-ethmoidal areas; polypoid membrane, both sphenoids, some pus and granulation tissue; right middle turbinate greatly thickened	Spheno-ethmoidal sinusitis, purulent, bilateral
49	Blindness, right eye, gradual return of vision, later failing; some vertigo and vomiting	1 year	Ethmoid walls and anterior wall of sphenoid very thin; membrane of sphenoid pale and edematous, vessels prominent	Spheno-ethmoidal sinusitis, hyperplastic, right; optic atrophy, right
50	Purulent postnasal dropping, cough; bioccipital headaches for 3 years; pain behind and above right eye for 8 months	4 years	All sinuses filled with polypi and pyogenic membrane	Pansinusitis, purulent
51	Constant frontal headaches, worse with frequent colds, not improved by treatment	5 years	Hyperplastic mucosa and pus, all posterior sinuses	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
52	Acute parenchymatous nephritis.....	Several years	Pus, both posterior sinuses, bone very hard to touch	Spheno-ethmoidal sinusitis, hyperplastic, bilateral; nephritis, advanced
53	Constant severe left frontal and occipital pain, relieved by nasal shrinkage	Several weeks	Thickened mucosa and pus, posterior ethmoidal and sphenoidal sinuses, left	Pansinusitis, purulent, left

54	Severe bioccipital headache; some gastric disturbance for several years	2 years	Pus, both spheno-ethmoidal areas; mucosa pyogenic, posterior sinuses	Spheno-ethmoidal sinusitis, purulent, bilateral
55	Postnasal mucoid discharge; anteronasal purulent discharge; bifrontal and occipital headache	4 years	Entire nasal mucosa thickened, mild hyperplasia of spheno-ethmoidal regions, vessels prominent; thickened mucosa of sphenoids and ethmoids, more so on left	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
56	Lower half headache, right, improved somewhat by treatment; failing vision, right eye; general anorexia for several years	1 year	Hyperplasia and pus, right sphenoid; polypoid degeneration of mucosa, left sphenoid	Spheno-ethmoidal sinusitis, purulent, bilateral; cataract, right
57	Asthma, spring and summer; sensitiveness to grasses; not relieved by pollen injection; difficult breathing through nose for several years	12 to 14 years	Polypi in olfactory fissures, bilateral; nasal mucosa pale, thickened, atonic	Spheno-ethmoidal sinusitis, hyperplastic, bilateral; asthma
58	Swelling and pain in right eye; eye grounds negative	3 days	Right side of nose swollen shut, pus in middle meatus and fronto-ethmoidal region, also right antrum	Acute pansinusitis, right; orbital cellulitis, right
59	Sepsis; chronic suppurative bronchitis.....	Several months	Pus from posterior sinuses.....	Spheno-ethmoidal sinusitis, purulent, bilateral; Maxillary sinusitis, purulent, bilateral
60	Headache, left frontal and occipital, with profuse purulent postnasal discharge; polyarthritis for 3 years	4 years	Lining of sphenoidal and ethmoidal sinuses greatly thickened, pus present	Pansinusitis, purulent, left
61	Bioccipital headaches, with mucoid postnasal dropping	Several years	Polypoid membrane, posterior sinuses.....	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
62	Constant purulent postnasal discharge; multiple arthritis, moderate	Several years	Polypoid membrane and pus, posterior sinuses	Spheno-ethmoidal sinusitis, purulent, bilateral; arthritis
63	Postnasal mucoid discharge.....	Several years	Polypoid membrane, mucopurulent discharge, posterior sinuses	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
64	Severe headaches, bioccipital; profuse postnasal mucopurulent discharge; asthma	Several years	Polypoid membrane and pus in posterior sinuses	Spheno-ethmoidal sinusitis, purulent, bilateral; maxillary sinusitis, purulent, bilateral
65	Severe bilateral occipital, parietal, temporal headache	2 years	Polypoid mucosa and pus, all posterior sinuses	Spheno-ethmoidal sinusitis, hyperplastic, bilateral
66	Iritis	2 months	Polypoid membrane, all posterior sinuses.....	Spheno-ethmoidal sinusitis, hyperplastic,
67	Atrophic rhinitis 36 years ago; severe bilateral headache (relieved somewhat 6 years ago by bilateral antral and intranasal frontal operation, recurring with added severity 4 months ago); profuse purulent discharge from all sinuses, with more severe headaches for 10 days	24 years	Extremely thickened, polypoid mucosa, slight pus, right sphenoid; large amount of pus, without thickening of mucosa, left sphenoid	bilateral, extreme; iritis
68	Partial blindness, left eye; paralysis of accommodation left eye; no headaches; small amount of postnasal pus for 2 months	6 weeks	Polypoid membrane, slight pus, left sphenoid; middle turbinate extremely large, cystic	Purulent pansinusitis, chronic
69	Asthma	Several years	Polypi and Thiersch grafts removed from both sphenoids	Spheno-ethmoidal sinusitis, hyperplastic, left
70	Arthritis (not relieved by tonsillectomy or teeth extractions); nephritis for several years	14 years	Pus and hyperplasia in both spheno-ethmoidal areas; polypoid mucosa, spheno-ethmoidal sinuses	Sphenoidal sinusitis, hyperplastic, bilateral Spheno-ethmoidal sinusitis, hyperplastic, bilateral; arthritis, chronic

verged, or in the spheno-ethmoidal region. Extensive true hyperplasia of a turbinate is, therefore, indicative of hyperplasia, or even of more extensive pathologic changes in the ethmoidal capsule and probably in the sphenoid as well (table 1). From the foregoing pathologic findings, it is seen that the changes in the tunica propria and the bone of the turbinate and ethmoid always go hand in hand.

Bone Changes.—These have a direct bearing on the foregoing vascular changes, so far as the extensive rarefaction and fibrosis are the results of diminished blood supply. As Carl Rohde says, after his exhaustive investigations on bone regeneration, "It is thus seen that through the entire question of bone regeneration the important and determining rôle of the vascular system runs like a thread of red. With its preservation and capacity for regeneration, the whole problem of regeneration stands and falls." The vascular changes are widespread and profound, and furthermore, are permanent. The bone changes assume a much greater significance when it is considered that but a small proportion of the diseased tissue was removed and that just as much, if not more, deep pathologic change exists in the remaining walls of the cells and sinuses. With this picture in mind, one can more readily grasp the reasons for the advanced eye changes and the extensive referred pains resulting from the pressure to which the respective nerves are subjected by the fibrosed tissue and bone which they traverse. By opening the cells and sinuses, the optimum condition for the improvement of this remaining pathologic change is established; namely, drainage aeration and accessibility to local treatment. Those patients have been considered as cured who have remained entirely free of symptoms. If symptoms have returned with acute exacerbations only, or have been markedly ameliorated, the patients have been designated as improved. "Not improved" designates no improvement, only very little, or that the patient has died. Many of these patients had other sinuses involved to a lesser degree than the posterior groups, and subsequently came to further operation. The patients from whom reports were not secured were, as a rule, from the outpatient department.

Comparing tables 1 and 2, it is readily apparent that the most marked changes have occurred in the tunica propria, with lesser affection of the epithelium and bone, and that the results of operation have had no relation to the character of the pathology. A similar pathologic picture has given rise to wide variation in local and general symptomatology; namely, headaches, asthma, nasal and postnasal discharge, failing vision, impaired hearing, herpes, arthritis and nephritis. In general, the duration of symptoms was in direct proportion to the fibrotic and bone changes. There was some tendency to polypoid changes of the epithelium and edema of the tunica in the asthmas and more acute conditions. The patients in whom there was immediate marked improvement

were those in whom there was either pus or polypoid tissue under pressure in the sinuses or cells. The more gradual recoveries showed that the extensive pathologic changes responded more or less rapidly to the regimen of aeration, drainage and local treatments, as gaged by the amelioration of symptoms. The nonrecoveries and deaths could not be explained from a pathologic point of view, as there was nothing characteristic about their pathologic pictures.

In considering the high percentage (74.3 per cent) of recoveries (table 3) following the removal of tissue in a state of advanced chronic

TABLE 3.—*Incidence and Results According to Age and Sex*

	Incidence							
	1 to 9 Years		10 to 19 Years		20 to 29 Years		30 to 39 Years	
Male	6	8%	2	3%	6	8%	2	3%
Female	..	..	1	1.5%	15	22%	12	18%

	Results															
	Cured	Improved	Not Improved	No Report	Cured	Improved	Not Improved	No Report	Cured	Improved	Not Improved	No Report	Cured	Improved	Not Improved	No Report
Male	1	1	3	1	1	..	1	..	1	3	D	1	..	2	..	..
Female	..	..	..	..	..	1	..	..	5	8	..	2	..	8	..	4

	Incidence							
	40 to 49 Years		50 to 59 Years		60 to 69 Years		Total	
Male	5	7%	2	3%	2	3%	25	35.7%
Female	11	15%	5	7%	1	1.5%	45	64.3%

	Results															
	Cured	Improved	Not Improved	No Report	Cured	Improved	Not Improved	No Report	Cured	Improved	Not Improved	No Report	Cured	Improved	Not Improved	No Report
Male	..	4	..	1	..	1	..	1	..	1	D	..	3 12%	12 48%	6 24%	4 16%
Female	2	9	..	..	1	2	1	1	..	1	..	..	8 17.7%	29 64.4%	1 2.2%	7 15.5%

fibrotic change, undergoing bone necrosis and rarefaction, there is good reason to assume that this type of tissue was largely responsible for the symptoms, especially the headaches, nasal discharge and arthritis, as present in 95 per cent of the cases.

The summary in table 3 emphasizes the fact that the incidence of operable spheno-ethmoidal sinusitis is almost twice as high among women as among men, and the percentage of cures and improvements is likewise much higher among the former (in the proportion of 40 to 1

among women, and only 2.5 to 1 among men). This could be explained partially by the fact that men are less prone to consult an otolaryngologist for a chronic nasal infection until it has become far advanced, and that women cooperate better in the matter of home and office treatment.

From figure 12, there seems to be no significant age incidence figures among men, except a slight preponderance between the ages of 20 and 30, and 40 and 50. Among women, 40 of the 64 per cent occurred between the ages of 20 and 40, with but a slight decrease between 40 and 50. There is a sharp rise between 20 and 30, and a more gradual decline at 50. From these figures, therefore, it may be concluded that women are relatively free from clinical sinus infection before and after the menstrual epoch. This naturally raises the question of the influence of the endocrine glands on sinus infections and the resultant syndromes, which we consider most pertinent and worthy of further investigation.

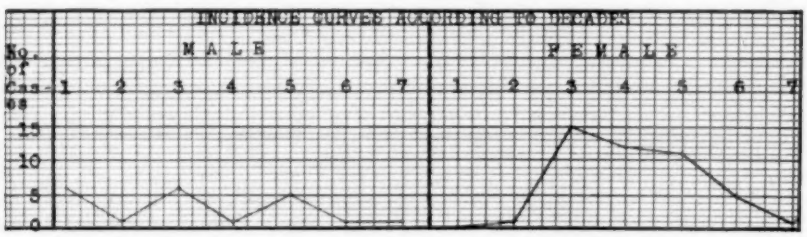

Fig. 12.—Incidence curve according to age and sex.

SUMMARY

1. The following microscopic changes were found in chronic hyperplastic spheno-ethmoidal sinusitis:

Epithelium.—Thickening; sloughing; polypoid degeneration; metaplasia.

Basement Membrane.—Thickening.

Tunica Propria.—Edema; round cell infiltration; fibrosis; dilatation or compression of glands; thickening of blood vessel walls.

Periosteum.—Thickening.

Bone.—Osteoblastic activity; osteoclastic activity; fibrosis; hyperostosis; osteomalacia; necrosis.

2. The following symptoms may be associated with the foregoing microscopic changes: Headache, anteronasal discharge, postnasal discharge, asthma, arthritis, failing vision, impaired hearing, and herpes of the second division of the fifth nerve.

3. There is no direct interrelation of individual microscopic findings, symptoms and results of operation.

4. The incidence of spheno-ethmoidal sinusitis is much greater among women than among men.

5. The percentage of good results is much higher among women than among men.

6. Chronic hyperplastic spheno-ethmoidal sinusitis is a distinct clinical entity, in which the operative results are attended with a high percentage of improvement or complete recovery (74.3 per cent).

Clinical Notes

EXTENSIVE LUNG ABSCESS INVOLVING THE ENTIRE RIGHT INFERIOR LOBE

Report of a Case Treated Successfully with Suction Through the Bronchoscope *

JOSEPH W. MILLER, M.D.
NEW YORK

M. R., a high school boy, aged 17, was admitted to the Beth David Hospital, on the service of Dr. G. Friedman, April 23, 1925. His complaint on admission was a cough with expectoration, fever, weakness and loss of weight for the past five weeks.

Previous History.—The patient was fairly well until Jan. 26, 1925, when he contracted a "cold" giving rise to fever, chills and cough which lasted about ten days. He improved so that he was able to go to school for one day; the next day he again had chills and high fever. A physician was called in, who diagnosed the case as right sided pneumonia. This lasted until March 8. During this time the patient coughed a good deal, lost 20 pounds (9 Kg.) in weight, and became very weak. He was sent to Lakewood where he stayed for five days, but was forced to return because of fever, cough and weakness.

March 13, his family physician was called again and a second time diagnosed the condition as right sided pneumonia. Since then, the patient's elevation of temperature was continuous and the cough apparently on the increase. Occasionally, the cough was so severe that he vomited. His appetite was gone, the bowels were constipated and weakness was marked.

It was learned that he had measles when 3 years of age, which resulted in a discharge from the left ear. This became chronic, and the ear was discharging for years when a mastoidectomy was performed at the age of 12. Two years later, a radical mastoidectomy was performed, but a dry ear was not obtained. His family history is not remarkable and throws no light on this subject.

Physical Examination.—The patient was poorly nourished, tall and thin. He was sitting up in bed, looking acutely ill, cyanotic and dyspneic. His left ear was draining slightly, the mastoid was covered with a healthy scar and no mastoid tenderness was present. The chest was symmetrical, with flatness and absence of fremitus at the right base posteriorly. Bronchial breathing and moist râles were audible at the base, especially near the spine. The heart was in the fifth space within the nipple line. No abnormal sounds or thrills were present, and the beats were regular and rapid. The abdomen was normal; no masses or organs were palpable. With the exception of clubbed fingers, the extremities were normal. The reflexes were hyperactive. The pulse was equal, regular and easily compressible.

A roentgenographic examination made April 23 disclosed clouding of the lower half of the right pulmonary field. The clouding was not homogenous. No fluid level was noted when the patient was in the upright position. Other roentgenographic examinations made up to May 9 showed similar readings.

The temperature on the day of admission to hospital was 103 F.; pulse 120, and respiration 24. The probable diagnosis was abscess of the lung—empyema?

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* Read before the Beth David Hospital Clinical Society, March 8, 1926.

* From the Otolaryngological Department, Beth David Hospital, service of Dr. Samuel J. Kopetzky.

April 25, some chest fluid was aspirated for examination. This was clear yellow, and showed no organism on smear; a culture was negative for tuberculosis. Differential count: Polymorphonuclears, 64 per cent; lymphocytes, 36 per cent.

April 27, expectoration was moderate and offensive, and separated into three layers. The temperature was typically septic, low in the morning and high at night. The sputum was negative for tuberculosis.

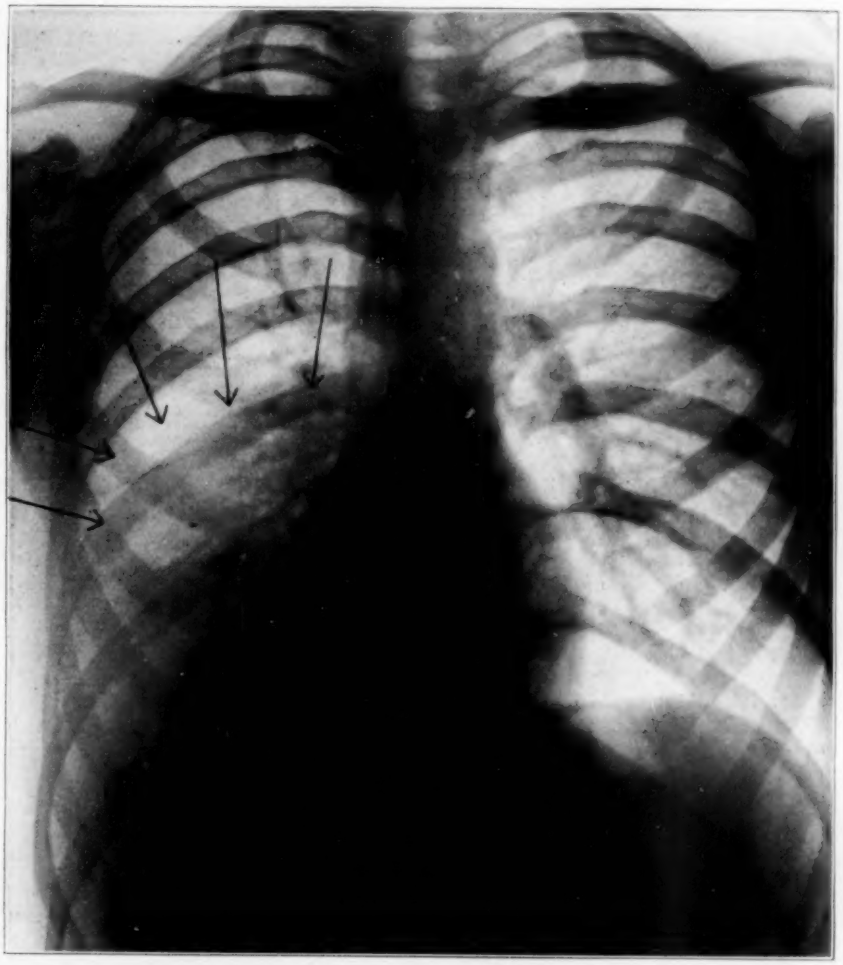


Fig. 1.—Clouding of the lower half of the right pulmonary field. The clouding is not homogenous. No fluid level was noted in the upright position. Other roentgenographic examinations made up to May 9, 1925, showed similar readings.

April 29, the temperature was still elevated to 102.8 F., the patient coughed less but looked much worse. He was put on 5 grains of guaiacol carbonate three times a day.

May 2, the temperature was septic, and the patient looked and felt badly. Amphoric breathing and râles at the right base, also râles and slight amphoric breathing at left base posteriorly, were present.

May 4, three spinal punctures in the seventh interspace disclosed no fluid.

May 7, the patient felt worse, coughed a little and expectorated less.

May 8, Dr. Miller, in consultation with Dr. Friedman, saw the patient, and it was decided to attack the lung abscess bronchoscopically.

May 9, the patient was actually moribund, and extremely emaciated; he was willing to undergo any treatment, and asked for relief.

Under local anesthesia, bronchoscopy was performed by me. About a half-hour before the bronchoscopy, the patient received a hypodermic injection of ¼ grain (0.01 Gm.) of morphine sulphate and $\frac{1}{150}$ grain of atropine sulphate. He was then taken to the operating room, and the anesthesia, composed of 10 per cent cocaine solution to which a few drops of epinephrine solution were added, was administered.

With the patient in a recumbent position, a Bruenings' type bronchoscope, size 10 mm., was passed along the trachea, and at the carina turned into the right main bronchus. The inner tube of the bronchoscope was now pushed gradually downward, and just as it passed the middle lobe bronchus, the tip sank into pus. This began to well up rapidly and was soon evacuated by suction. Almost a pint of greenish gray purulent material was obtained. No irrigation was used. As there was no more pus forthcoming the bronchoscope was removed and the patient was returned to bed.

Once in bed he felt much better, and although tired from the ordeal and breathing rapidly, he was anxious to know when bronchoscopy would be performed again.

A roentgenogram of the chest following the bronchoscopy revealed a patch of clouding of the right base, sharply demarcated above, and within this area there were numerous small foci of aeration. There was no fluid level in the lateral recumbent position. Above the area of consolidation there was an area of pneumothorax.

This report somewhat startled the physicians, and bronchoscopies were discontinued. In spite of the pneumothorax, the contributary cause of which I could not figure out, the patient's breathing was less labored, his cyanosis was almost gone and subjectively he felt much better and was more active. But the lung signs soon changed; the symptoms of pneumothorax gradually disappeared, while such signs as flatness, bronchial breathing and numerous moist râles, best heard in the right base posteriorly, once again dominated the picture. A roentgenogram of the chest taken on May 28, showed a large patch of clouding occupying the lower and inner portion of the right pulmonary field. Within these portions there were several small foci of aeration. The extent was about as previously reported. Another roentgenogram, taken June 3, revealed a patch of clouding in the lower third of the pulmonary field. There was no fluid level with the patient in the upright position. The extent of the process was not diminished since the original examination.

These observations, as well as the physical signs, demonstrated that the pneumothorax had disappeared, but that the extent of the lung involvement remained the same. The patient, however, felt much better.

June 3, râles over the cavity were fewer and small. The cavity seemed clearer. The patient was better subjectively, and felt more active.

June 4, the temperature was 102 F.; subjectively, the patient felt well. The cavity seemed clear on physical signs; only very few râles were heard. Cavernous breathing and also impaired resonance were still heard over the cavity. These observations, with only a little change here and there, were noted on the chart daily, and although the patient felt much better subjectively, he did not gain in weight or in strength. The physicians then decided to have another bronchoscopy performed; therefore, June 8, with the patient under local anesthesia, I performed the operation and about a half a pint of pus

was removed by suction. The following day the patient looked and felt better, and June 12 he said the sputum was no longer offensive to him. He coughed up easily a large quantity of yellow mucopurulent sputum in twenty-four hours. The area of dulness on the right side posteriorly took a somewhat resonant note. The appetite improved, and the weight increased to 100 pounds (45 Kg.), a net gain of 6 pounds since the first bronchoscopy.

Permission was now given by the patient's physicians to perform a bronchoscopy every week. This was carried out five times at weekly intervals, and a total of seven bronchoscopies were performed. The patient improved more each time. A roentgenogram of the chest, July 13, as compared with the previous roentgenograms, showed a diminution in density of the involved area. The patient left the hospital on August 2, much improved and gaining in

Fig. 2.—The final roentgenogram made Oct. 6, 1925, showing a fibrosis gradually obliterating the lesion.

weight and strength. He was referred to the outpatient department where he was observed from time to time; although he looked and felt well, he complained that he expectorated quantities of purulent material once or twice daily. This annoying condition gave me the idea of how best to produce a fibrosis of the involved area, so the patient was referred to the deep therapy department of the Beth Israel Hospital, service of Dr. Gustav Bucky. There he received 6 irradiations within two months, which gradually reduced the sputum evacuations at first to three times weekly, then to once a week and finally stopped them altogether.

Roentgenologic examination of the chest made during the various periods while receiving deep therapy are very interesting, and show a fibrosis gradually obliterating the lesion as time went on.

COMMENT

When this paper was written, the patient was in excellent health; he no longer coughed or expectorated purulent material, and he had gained 48 pounds in weight.

268 East Broadway.

A NEW TONSIL NEEDLE HOLDER AND ARTERY HEMOSTAT

MORRIS A. WEINSTEIN, M.D.
PHILADELPHIA

In presenting this new instrument, I should like to stress its significant advantages and its distinguishing features.

The instrument is so constructed that its distal end slopes into a shallow semicircle, with tapered end instead of a pointed tip. The reason for this is to permit the convexity of the needle holder to fit into the concavity of the tonsillar fossa, to allow penetration behind the posterior pillar for almost its entire area, and, by using the shank as a fulcrum, to elevate and approximate the posterior pillar toward the anterior pillar.

The semicircular end is serrated on its medial surface, thus providing grooves for the needle, which is inserted with its tip always protruding from the convex aspect of the arch.

The catgut strand is knotted near the eye of the needle, which is always advisable in order to prevent the needle from slipping out of the grasp after its tip has been secured and the holder removed.

Fig. 1.—Position of needle in the holder.

The instrument is of exceptional service when suturing low down near the base of the tongue, or when the posterior pillar, the bottom of the tonsillar fossa and the anterior pillar are sutured together with one sweep.

The shank of the needle holder remains in the center of the mouth while turning on its vertical axis, and leaves the operator a clear view of his working field—a no mean advantage when operating in so small and so limited an area.

While it is advisable to use the hand corresponding with the side to be sutured, it is of distinct advantage to step to the back of the patient's head when the left side has to be sutured, as the operator is prone to make use of his right hand. In this way, it is astonishing to note how low down near the base of the tongue one may reach.

The tip may be used as an artery hemostat to catch a bleeding point, or the entire length of the convex arch may grasp a larger area when a venous oozing is encountered. It can also be used with advantage as a needle holder in cleft palate work, the convexity of the semicircular distal end fitting into the concavity of the palate.

I have used this needle holder on an extensive series of subjects with great satisfaction, and the same gratifying results were reported to me by several of my colleagues who have used it in their operative work.

1309 Spruce Street.

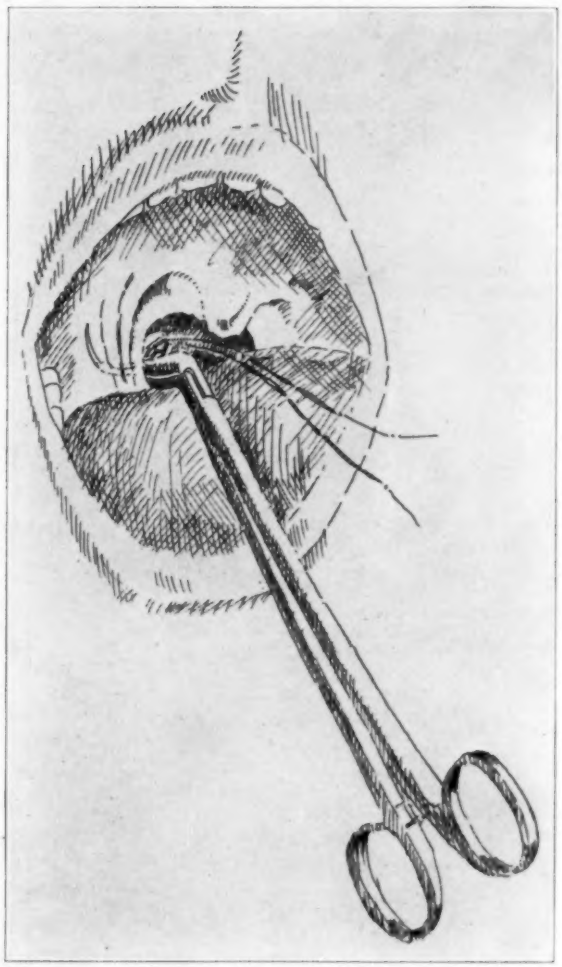


Fig. 2.—Needle going through posterior pillar, base of tonsillar fossa and anterior pillar.

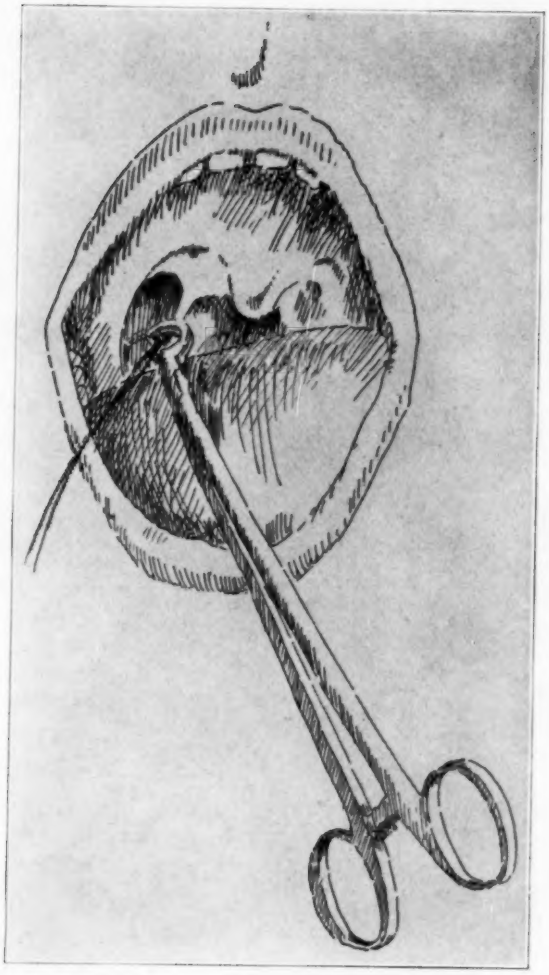


Fig. 3.—Suturing inside the tonsillar fossa.

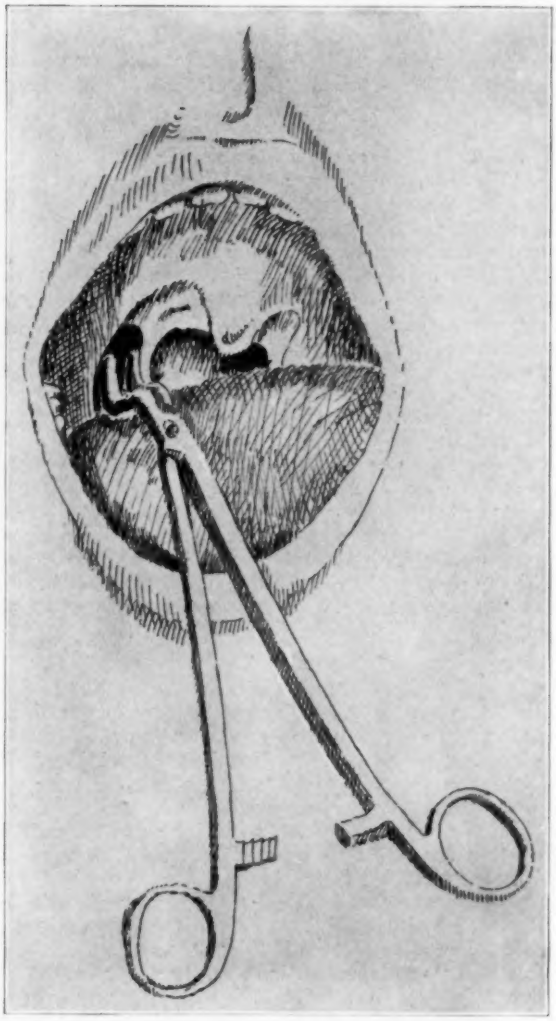


Fig. 4.—Grasping a large bleeding surface (hemostatic action).

A NEW PROCEDURE FOR THE CLOSURE OF NONSPECIFIC PERFORATIONS OF THE NASAL SEPTUM

M. A. LISCHKOFF, M.D., AND C. J. HEINBERG, M.D.
PENSACOLA, FLA.

A recent experience in the closure of a small whistling perforation of the septum is the basis of this paper.

A woman, aged 50, had a small anterior perforation of the septum, approximately 5 mm. in diameter. She had frequently picked her nose, causing crusts to form and recur. As the crusts would form, she would pick them away. This she continued to do until she had caused a true perforation.

Because the insertion of ivory into the submucous space of the septum has been of value in the successful treatment of atrophic rhinitis, we considered its use in this case. We therefore pursued the following plan of procedure: An incision of the anterior mucous membrane, which is usually used in submucous resection of the septum, was made. The mucous membrane was carefully dissected away from the cartilage. At the same time, the edges of the perforations were freshened. The entire cartilage was removed, and a thin, wafer-like piece of ivory was inserted into the submucous space. This piece of ivory rested on the maxillary ridge, and extended to the bony septum posteriorly. The incision was sutured, and in this manner the septal perforation was completely obliterated, leaving a small area of exposed ivory on each side. In considering this procedure, we felt that the surface of the ivory might act as a bridge over which the growth of epithelial cells would take place. This might be helped by a stimulating ointment, such as scarlet red.

So far as the operation was concerned, the patient recovered, with no ill effects. Postoperative care consisted in the use of scarlet red ointment, cleansing with a mild alkaline spray, and the occasional use of a weak solution of nitrate of silver. When last seen by us, months after the operation, the surface of the ivory was beginning to be covered by a new growth of epithelium. Of course the whistling was no longer present, and there was much less crusting around the edges of the implant. A roentgenogram showed the implant in the original position.

COMMENT

The closure of septal perforations always holds the interest of the rhinologist. This, no doubt, is because of the difficulty encountered in securing satisfactory results when we attempt to overcome this condition. In most instances, patients are advised to disregard the perforation if it is not troubling them. The perforations for which, as a rule, the patients seek repair are those that cause an annoying whistling sound. In this particular instance it was the annoying sound and fear of a possible malignancy, resulting from the perforation which brought the patient to us. She was assured that no such thing would occur, but insisted on having something done.

The sliding and the hinged flap methods of closure are difficult and frequently unsuccessful. It was for this additional reason that we decided on the procedure mentioned. The simplicity of this procedure, and the apparently satisfactory result after a period of months, leads us to believe that the implanation of ivory for septal perforation is worthy of consideration.

404 Blount Building.

Abstracts from Current Literature

Ear

Surgery of the Middle Cranial Fossa. Oscar Beck, Monatschr. f. Ohrenh. **3**:199 (March) 1926.

Complications of the middle cranial fossa in the course of acute or chronic middle ear suppurations extend usually through the tegmen tympani or antri. The infection may travel quite deeply as far as the foramen ovale and spinosum, and anteriorly as far as the fissura orbitalis. The technic of the operations in adequately exposing this vicinity has been simple enough, but the cosmetic results were at times disfiguring. Splitting of the temporal muscle produced a sunken scar anterior to the auricle and which by its traction pulled the auricle anteriorly. Extensive cicatrization resulted, too, between the exposed dura of the middle fossa (after wide extirpation of the skull wall), and the skin.

Beck cites five patients operated on according to the technic introduced by Neumann. The regular crescentic incision is made behind the auricle and the mastoid operation completed. The crescentic incision is then prolonged superiorly and anteriorly into the hairy area of the skull. The temporal fascia is carefully dissected from the zygomatic arch, and the temporal muscle with its fascial sheath intact is loosened with the pericranium. The temporal muscle can now be easily retracted anteriorly.

Healing results without a retracting scar. When the temporal muscle, with its sheath intact, is replaced in its fossa, it covers the gap in the cranial bone and the cutaneous scar is flat and smooth. The course of the incision is schematically illustrated on a caricature head of Mussolini.

Glowacki, Detroit.

Indications for Opening the Mastoid Process in Acute Middle Ear Suppuration. Ove Strandberg and Aage Plum, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:424 (April 27) 1926.

The authors quote from the literature indications for the mastoid operation. In their material they found an epidural abscess twice during the first four days of the suppuration, once in connection with a perisinus abscess and collateral meningitis; perisinusitis in the first three days and pachymeningitis externa in the first four days. The authors mention that an osteitis in the mastoid process can be assumed if, in spite of the sufficient drainage through the perforation in the drum membrane and treatment with warm poultices, the following symptoms persist:

1. Infiltration or tenderness increase
2. Infiltration of the inner portion of the meatus increases or develops
3. The temperature decreases or rises
4. Pain in the ear persists or sets in
5. The drum membrane remains bulging, or remains succulent
6. The hearing plainly decreases
7. Symptoms of labyrinthian irritation appear
8. Half sided headache appears

9. A discrepancy between temperature and pulse appears, or the pulse becomes irregular or of bad quality

10. Albuminuria sets in

11. The general condition becomes plainly affected.

It is very difficult to decide whether a mastoid operation should be performed when the labyrinth is in a state of irritation. Whatever one does is fraught with danger. If one waits, the inflammation may progress, the labyrinth may be destroyed and meningeal complications may ensue. If one operates, an infection may be carried into the labyrinth. The authors once encountered such a labyrinth irritation one and one-half days after the first vague symptoms of the ear trouble, but the nystagmus disappeared after the paracentesis. The patient was cured without further interference. Spontaneous nystagmus to the diseased side was noticed four times during the first two or three days after paracentesis. Mainly for this reason the mastoid was opened. All these patients were cured with normal hearing. Expectative treatment was contraindicated. Patients of this nature are operated on with great care. Only freshly sharpened chisels are used in order to traumatize as little as possible.

THE NATURE OF HARDNESS OF HEARING DURING PREGNANCY. O. MUCK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:441 (April 27) 1926.

With the aid of the epinephrine probe experiment, the tonus of the sympathicus can be established. Its hyperexcitability is shown by the white stripe which is not found in healthy people. This hypertonia of the sympathicus of the vessels of the brain is found permanently and periodically, especially in hemicrania and epilepsy. The sympathicohypertonia of the vessels is very frequent (72 per cent) in the second month of pregnancy. The hypertonia curve drops to 28 per cent in the third month, and to 22 per cent in the fourth month. This curve points to certain interrelations between "humoral processes" and the tonus of the sympathicus of the head, taking into consideration the toxicoses of pregnancy (Seitz). The gynecologist and rhinologist should cooperate concerning the toxicoses of early and late pregnancy. It is absolutely essential to discover whether a continuous hypertonia exists in the sympathicus of the head, or whether it exists only during some months in a pregnant woman. A uniformity of the conception does not exist to decide whether a pregnancy should be interrupted to save the hearing endangered by pregnancy. Yet it has been shown that the prognosis concerning hearing is bad if the hearing disturbance occurs during the first days of pregnancy and that an interruption of pregnancy (when that is the cause) reestablishes the hearing or prevents a further decrease. Endocrine disturbances must also be considered (Frey and Orzechowsky, Voss, Denker and Leicher), whether true otosclerosis is present, or whether a hearing disturbance occurs during pregnancy. If an inner ear type of hardness of hearing occurs during pregnancy, and if the epinephrine probe experiment shows a sympathicohypertonia of the vessels of the brain, a labyrinthopathia vasogenica e graviditate is present. A decrease of hearing in pregnancy in true otosclerosis, with symptoms of irritation of the vestibularis and cochlearis, must be looked on as an angioneurotic attack caused by pregnancy, because the labyrinthian disturbance sets in suddenly. Muck reports a case in point. The patient, aged 35, the wife of a physician stated: "During the first month of the third pregnancy, in 1923, a very violent feeling of dizziness occurred, so that I could not stand up, and I had to lie down for one hour

before I could walk straight. I felt very nauseated. A strong tinnitus existed. The hearing had decreased for about three weeks. A decrease of hearing for high tones persisted." The status of the hearing in 1926: The drum membranes did not show any pathologic changes. Both tubes were free.

right	left
C_2—7	—9.
C_4—6	—9.
C_1 =	=
Rinne +	+
Whispered voice 10 m.	10 m

The Weber reaction was undecided, the labyrinth reacted normally to irritation. Bone conduction was not essentially shortened. In this instance, a slight hardness of hearing was present during the first month of pregnancy.

THE PATHOHISTOLOGIC CHANGES OF THE HEARING ORGAN IN TYPHUS. N. T. POPOFF, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:477 (April 27) 1926.

The author describes the various changes in the different parts of the hearing organ, including the eighth nerve and its origin, as observed in Moscow. A temporary disturbance of hearing or a more or less pronounced hardness of hearing can be caused by changes of the drum membrane by hyperemia, hemorrhage and desquamation of the epithelium. Pronounced changes can occur in the middle ear, in its mucous membrane and the underlying tissues. The joints of the ossicles can be affected and the muscles of the middle ear may degenerate. While Corti's organ shows many changes under the microscope, it is especially the vessel system of the nerve which reacts to the virus by a "destructive thrombovasculitis" with dilation of the vessel, stasis, hemorrhages and thrombi. Hemorrhages into the nerve were so violent in spots that whole bundles of nerve fibers were torn. The surrounding tissue shows infiltration, the cell elements of which consist of cells resembling lymphocytes, plasma cells and cells of the polyblast type. Not infrequently macrophages are also present. Characteristic for typhus exanthematicus are peculiar nodules, which are found in great numbers in the eighth nerve and which extend along the fibers and across the trunk of the nerve. Concerning stationary total deafness, the injury of Corti's organ and of the acoustic nerve must be considered. Unsurmountable technical difficulties prevented the study of processes of regeneration and the topography of the special elements, yet the investigations of the author point to the fact that Corti's organ is not affected in all its parts without hope of restitution. The main weight of the condition of the hearing organ falls on the peripheral tract of the cochlear nerve, as is shown by observing the changes of the peripheral portion of the eighth nerve from the nuclei to the entrance in the lamina spiralis ossea. The degree of the condition decides the prognosis of the hardness of hearing and the therapy.

AMBERG, Detroit.

Pharynx

NECROSIS OF THE PALATE AS PRIMARY SYMPTOM OF A MYELOBLAST LEUKEMIA AND FINDINGS IN THE THROAT IN A CASE OF ALEUKEMIC LYMPHADENOSIS. ELSE LEVY, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:468 (April 27) 1926.

Rudolph Virchow has described the diseases of the throat and pharynx in his fundamental contributions concerning leukemia. Two cases are reported. Both came under medical care when difficulties on the part of the throat, and

of the mouth, respectively, set in. The first case (of Minnigerode which is exhaustively reported by E. Levy), also showed other symptoms referring to leukemia. In Levy's patient, all symptoms referring to leukemia were absent. Therefore, it is necessary that one must think of a disease of the hematopoietic apparatus if only one symptom is present, namely, glandular swellings, swelling of the adenoid tissue, necroses of nuclear etiology or a peculiar discoloration of the face (pale yellowish). Under all circumstances, a blood test should be made. Not only the blood elements must be counted, but their exact cytologic quality must be established.

AMBERG, Detroit.

Larynx

SOME DISORDERS OF THE LARYNX. V. E. NEGUS, Lancet **2**:581 (Sept. 19) 1925.

The normal movements of the larynx and glottis are discussed, and this paper is an attempt to explain these movements. A clear exposition is given of the gaseous exchange which takes place in the lungs during respiration. It is pointed out that the glottis is a choked point on the airway at all times, so it causes a slow and gradual filling and emptying of the lungs. Even during powerful inspiration the glottis, though wider, still maintains the negative presure which is normally present at the beginning of inspiration. This negative pressure causes an increased flow of blood through the lungs at this moment, and as the alveolar air is now lower in carbon dioxide, the blood gives up its excess. During expiration, when the carbon dioxide in the alveolar air is relatively higher, there is less blood flowing through the lungs, thus the carbon dioxide content of the blood is kept relatively constant.

From the study of comparative anatomy the author believes that the obstruction offered by the glottis during expiration, may have something to do with the distribution of air to the distally situated alveoli. The reason for the priority of abductor paralysis resulting from injury of the recurrent laryngeal nerve is ascribed to the fact that the adductor group of muscles is older in evolution. In certain lung fish there are only the sphincter like adductors. The abductors are only found in more highly developed types.

The effects of different types of laryngeal obstruction are considered and their influence on the circulation is explained. The physiologic factor of glottic obstruction is suddenly removed by tracheotomy, so the carbon dioxide content of the alveolar air and circulation falls below normal and the patient may die of apnea from lack of respiratory stimulus. The carbon dioxide content of the alveolar air should be lowered gradually, which can be provided for by adding carbon dioxide to the inspired air. Then follows a more detailed account of the effects of tracheotomy and laryngectomy.

In considering anastamosis of the recurrent laryngeal nerve, the author believes that though the result is admirable for ordinary purposes, the fine adjustment during inspiration and expiration is lost. The reasons for this belief are given in detail.

MCNALLY, Montreal.

THE REMOVAL OF THE EPIGLOTTIS. W. FREUDENTHAL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:486 (April 27) 1926.

The author claims that a cure after removal of the epiglottis was rare. He bases his judgment on the observation of thousands of cases of tuberculosis of the larynx which he has seen during thirty-one years as chief of the laryngologic department of a large hospital. Formerly the operation was per-

formed more frequently, but experience has taught that even the most simple operation on the larynx of people suffering from tuberculosis may be followed by disagreeable results (severe hemorrhage of the lung, etc). Commenting on Buech's article in the *Ztschr. f. Hals-, Nasen- u. Ohrenh.* in which he claimed that care in indication was partly responsible for good results, Freudenthal says that Buech neglects to mention the indications. Freudenthal acknowledges only two, first, increasing dyspnea or presence of the same, when tracheotomy can be avoided by the more simple operation of resection of the epiglottis. Uncomplicated infiltration of the epiglottis is rare. In most cases a perichondritis of one or both arytenoid cartilages, or infiltrations of other parts of the larynx necessitate a tracheotomy; second, if painful ulcers can be made more easily accessible in dysphagia which cannot be combated otherwise. The author prefers suspension laryngoscopy for the operation except in cases of far advanced tuberculosis of the lung (cavities).

AMBERG, Detroit.

Nose

SYMPATHETIC OPHTHALMIA CURED AFTER EXENTERATION OF THE NASAL ACCESSORY SINUSES. BEN WITT KEY, Am. J. Ophth. **9**:574-583 (Aug.) 1926.

The author reports a case of a man, aged 48 years, who, May 26, 1925, was struck in the eye by a small piece of metal. Four days later the foreign body was removed by a magnet. July 15, the injured eye was very sensitive to light with active conjunctival and iritic reaction; the globe penetrated at the temporal limbus and the iris prolapsed; the cornea was slightly hazy with a few spots on Descemet's membrane; the iris was congested and sluggish in reaction; the vision was 20/20—. The left eye was very sensitive to light but only mildly injected; the cornea was clear except for a few deposits on Descemet's membrane; the iris was very slightly congested and partly dilated; the pupil was large, round and regular, and evidently sluggish from the effect of atropine; the pupillary space was clear, and reflected a clear and transparent lens; the vision was 20/20.

Intensive treatment was administered, consisting of hot bathing and atrophine, catharsis, mercurial inunctions, sodium salicylate and antidiphtheritic serum. Complete laboratory examination was negative. The opinion after consultation with a number of consultants was about equally divided for and against enucleation.

In the next four weeks both eyes became gradually more actively involved, and the injured eye took on a characteristic low-grade form of acute uveitis, later hypertension and finally detachment of the retina and complete loss of vision. The sympathizing eye at the same time showed the classical low grade plastic uveitis of sympathetic ophthalmia. The iris was discolored and thickened, the pupillary space was partly filled with thin exudate, the vitreous was very cloudy, the tension was very soft and vision was reduced to 5/200. At this time the injured eye (the right) was removed.

Two days after enucleation the patient was first seen by the author, and an examination of the nasal sinuses was made. Dr. George Dixon reported the following: "Frontals well developed; left ethmoids cloudy; left antrum occluded; left side of nose almost occluded. Nasal septum deflected to the left. Right sphenoid and antrum fairly clear." The right antrum was punctured and the ethmoids were opened and drained. Both were found to be full of pus, the culture of which showed the pneumococcus capsulatus mucosa. By sixty hours later the vision of the left eye had improved to 20/200.

This improvement lasted for about five days when there was a gradual return of the eye to its previous condition and vision was reduced to 3/200. Again the sinuses were explored, but more thoroughly. The ethmoids were found to be extensively involved and were partially exenterated. Within forty-eight hours the eye was definitely improved and the vision was 20/70. Forty-eight hours later the vision was 20/70 with gradual daily improvement in the appearance of the eye over a period of about a week. But now for the third time, it could be observed that the eye was again losing ground, and in the course of a few days the vision was again reduced to 5/200. For the third time the sinuses were explored, both sphenoids as well as the ethmoids being found to be filled with pus. Following this extensive operation the eye again responded and in two days the vision was 20/200. In the course of ten days more the fundus for the first time could be indistinctly made out and the vision was 20/30. In a few more days the eye was almost entirely quiet, very slight iritic reaction and vision 20/20—2 letters.

Three weeks after the last intranasal operation, the patient had left acute otitis media followed by acute mastoiditis. During the development of the acute mastoiditis, the left eye began to show the effects of this renewed toxemia and the vision became gradually reduced to 20/50, with corresponding uveal reaction. The mastoid was operated on, the only complication being a left facial paralysis. The eye rapidly became quiet. The cornea, aqueous and lens became clear. The iris remained pale and atrophic with numerous synechiae well shown by the slit lamp, and in addition a few small spots of exudate were present on Descemet's membrane. On examination by the slit lamp the aqueous beam was accentuated, and a moderate number of fine particles were observed in the aqueous stream. There was evidence of pigment derangement on the posterior surface of the cornea in the aqueous, iris crypts and on the lens capsule. The pupil was irregular in outline and responded feebly to direct light. The ophthalmoscopic examination showed a few small floating particles in the vitreous but the fundus was normal. Intra-ocular tension was normal and the vision with correction was 20/20.

The author goes into detail about the microscopic examination of the enucleated eyeball, giving photomicrographs and the results of the histologic examination.

In conclusion, he feels that the microscopic picture showed the typical changes recognized as occurring in cases of true sympathetic ophthalmia. His opinion that this was a case of sympathetic ophthalmia was concurred in by eight or ten eminent ophthalmologists. The author asks if this sympathetic ophthalmia then was due to any cause other than the pansinusitis, and was it reasonable that by mere coincidence a recrudescence in this sympathetic process could take place four times, each followed by final cure of such an eye having at one time vision of fingers at three feet. The author states that medicinal treatment had failed over a period of one month, during the steady march of the disease, from almost normal vision in each eye to the complete destruction of the injured eye, the sympathizing eye being reduced to a visual acuity of 5/200. The rapidity with which the eye improved after exenteration of the sinuses was significant. Although the possibility of a retrobulbar neuritis as contributing to the loss and rapid return of vision cannot be ruled out, it does not alter the fact that the clinical picture of the uveitis was sufficient to account for the great reduction of vision.

The author believes he is justified in stating at this time that it seems reasonable to assume that true sympathetic ophthalmia may have its origin or is excited by a focus of infection; which, by its toxins, or bacteria in the circulation,

affects the injured eye in such a manner as to aid in similarly causing like changes in the fellow eye directly and in association with it. By "association with it" he refers to the allergic state attributed to uveal pigment hypersensitivity, occurring as the result of injury or other destructive changes in the uvea of the sympathogenic eye as worked out by Woods.

The author states it seems reasonable to assume that sympathetic ophthalmia is a specific type of uveitis due to a focus of infection or systemic disease either exogenous or endogenous, just as other types of uveitis have their origin. But the clinical picture and the destructive nature of the sympathetic type are due to the pigment hypersensitiveness and hypersusceptibility of both eyes, incident to uveal destructive changes from injury to the globe. The therapeutic indication, therefore, is the search for and elimination of the focus of infection.

H. H. Vail, Cincinnati.

Congenital Absence of the Middle Turbinated Bone Associated with Precocious Ossification of the Limb Bones in a Stillborn Female. H. A. Harris, J. Anat. **60**:148 (Jan.) 1926.

This is a report of a stillborn female child, of breech presentation. It showed considerable cephalhematoma. A most advanced degree of centers of ossification were encountered in the limbs. There was complete absence of the right middle turbinate. The other nasal structures were normal except for segmentation of the lamina papyracea on the left side. Zuckerkandal reports the absence of the middle turbinate in 13 per cent of a series of 267 sagittal sections of the skull. Comparative anatomy shows remarkable variation in the number and shape of the turbinates. The nasal septum, inferior and superior turbinates do not show such variation as does the middle turbinate. Le Double says he knows of no proved case of absence of the inferior turbinate bone. It has been remarked that nasal polypi are mostly associated with the middle turbinate, and that the part subject to the largest amount of variation is also most subject to disease.

Recovery from Tubal Deafness Caused by a Sarcoma. Samuel Lodge and William Oliver Lodge, Brit. M. J. **2**:826 (May) 1926.

After removing a sarcoma from the soft palate, a unilateral deafness of eight years standing cleared up. The question is raised as to what is the pathologic changes in the ear in these cases. In three cases of obliteration of the eustachian tube from tumors, Brock found that there was a serous exudate in the tympanic cavity attributable to "hydrops ex vacuo" rather than to inflammation. Karl Beck obliterated the eustachian tube in dogs by cauterization. He found hypertrophy and cellular infiltration of the mucous membrane, and a sterile exudate. According to Habermann, this condition differs from chronic catarrh of the middle ear, in which the proliferative changes in the tympanic mucosa are identical with those of hypertrophic rhinitis. In the case here reported, there were no subjective symptoms of fluid in the tympanic cavity nor could fluid be seen through the drum.

Chronic Nasal Discharge in Infants. A. Brown Kelly, Lancet **2**:1083 (Nov. 21) 1925.

There are three varieties of acute rhinitis from which a new-born infant may suffer: (*a*) Coryza neonatorum simplex, which resembles the ordinary head cold of the adult; (*b*) coryza neonatorum blenorrhagica, infection usually

resulting from a leukorrheal or gonorrheal discharge in the mother's vagina at the time of birth; (*c*) coryza neonatorum syphilitica, which is a manifestation of secondary inherited syphilis. The differential diagnosis of these three types is discussed. Owing to the fact that children do not readily adopt mouth breathing, there is a tendency to suffocation when the nose is blocked. Sucking is also interfered with because the child must stop at frequent intervals to take a breath. Early and careful treatment of the symptoms and cause is most important. If nasal obstruction is marked a little epinephrine (1:10,000) may be painted in each nostril. Any crusts must be softened and removed. The catarrh is best treated by silver preparations. In the syphilitic subjects, a paint of colloidal mercury or an insufflation of calomel and sugar (1:25) may be employed.

Nasal suppuration is occasionally caused by an osteomyelitis of the superior maxilla. It is usually an empyema of a dental sac, the infection being hematogenous. This must not be mistaken for an antral empyema. A tooth projecting through the jaw should be removed, and thorough search made for sequestrums.

Chronic Nasal Discharge and Sinusitis in Children. A. Brown Kelly, Lancet **2**:1132 (Nov. 28) 1925.

A foreign body may be the cause of a chronic nasal discharge, and if this is suspected in a particular case, great care must be exercised in manipulation. If the child is unmanageable it should be anesthetized. Before the anesthetic some epinephrine (1:10,000) may be sprayed in the nose to facilitate a more thorough examination.

Chronic rhinitis comprises two classes of cases: (1) When the lining membrane of the nose has undergone inflammatory thickening, the more common causes being frequent colds, obstruction, septal deviations, etc.; (2) hypersecretion from the more general causes, tuberculosis, intestinal toxemia, nephritis, etc. Diseased tonsils and adenoids are frequently the cause of a chronic rhinitis and their removal will sometimes result in a cure of the nasal condition. If there is marked swelling of the inferior turbinates, the hypertrophy may persist after the operation and even require judicious removal with the scissors. The most intractable discharge is that which follows scarlet fever; it is benefited by mild cauterization with a solution of chromic acid (from 10 to 50 per cent). If there is any vasomotor disturbance the protein sensitization should be investigated. With reference to the general health and surroundings of the patient, a change is often followed by a cure. Sinusitis, particularly of antral and ethmoidal origin may be present even in early infancy. They should be carefully investigated by roentgen ray, and if possible by exploratory lavage. The antrum can be reached through the inferior meatus in children over 8 years of age. The first in the treatment is the removal of the tonsils and adenoids. Instillations into the nose of 10 per cent argyrol are beneficial.

Lupus of the Nose and Upper Air Passages Treated by Radium. H. Mortimer Wharry and Oskar Teichman, Lancet **2**:1275 (Dec. 19) 1925.

Lupus affects the mucous lining of the nose, mouth and upper air passages quite as often as the skin of the face and neck. It attacks both sexes equally; in a greater number the subjects are about the age of 20 and are of the lower middle class.

These authors recognize four types of lesions: (*a*) Granulations; (*b*) vegetations or proliferations; (*c*) tumors; (*d*) ulcerations. Of these types the first three are particularly favorable for radium treatment. The fourth type does not yield so readily because there is less granulation tissue formed than in the other cases. The different modes of spread are discussed. Though these patients may be suffering from a very extensive lesion, they are not definitely ill. The local symptoms vary with the site of the lesion. There is a divergence of opinion about the prognosis, but the authors believe that in the majority of cases, if properly treated, there is a definite disappearance of the disease, and even the cases of type four show marked improvement.

The general treatment is similar to that of tuberculosis. For local treatment in slight cases, when the disease is readily accessible, the galvanocautery is used with very good results. In all other cases radium treatment is confidently recommended. These authors urge early recognition and treatment of the disease. The radium in lethal doses destroys living tissue, and in stimulating doses it accelerates the formation of fibroblasts, and so assists the maturing of granulation tissue. In practical dosage it inhibits but does not kill the tubercle bacillus. The scar tissue following the use of radium is soft and pliable, so that the function of the part is very little interfered with as a rule.

A clear account is given of the different radium rays, and it is pointed out that the beta rays are destructive and cause a surface reaction. The gamma rays are penetrating and stimulate the fibroblasts. By varying the composition and thickness of the container, the kind and the desired amount of the rays can be regulated to suit each case. There is an appendix reported of patients recently treated who showed very satisfactory results.

McNALLY, Montreal.

TREATMENT OF OZENA. E. LEICHSENRING, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:422 (April 27) 1926.

Leichsenring employed Soyka's method of irritation. It proved to be a practical impossibility to use the tampon therapy with the patients belonging to the sick benefit association, because they could not afford the time. Therefore he recommended to his patients a brand of snuff. He permits the patients to wash out their noses, and sometimes he applies acriflavine powder twice by insufflation in the nose. Leichsenring uses this powder because in ozena a microbe is frequently found which can scarcely be differentiated from the diphtheria bacillus. The treatment lasted sometimes for several months, but patients could be observed who considered themselves cured after eight days. Leichsenring claims quite favorable results concerning secretion, odor and also atrophy.

THE EPINEPHRINE PROBE TEST OF MUCK. E. BUECH, Ztschr. f. Hals.-, Nasen- u. Ohrenh. **14**:446 (April 27) 1926.

Buech found that the epinephrine probe test did not show a perfect result in more than thirty patients. The mucous membrane did not take up the epinephrine. On that day the weather was extremely foggy, and the mucous membrane was covered with a layer of a glassy, impenetrable mucus which was scarcely visible. A congestive hyperemia, as is sometimes caused by wearing too narrow a collar, is disturbing. Furthermore, in old people, the senile mucous membrane shows a scarcity of cavernous tissue. For this reason, the reaction of the mucous membrane of the turbinate is interfered with. After

the epinephrine pallor has appeared, Buech carries a blunt probe five or six times slowly over the most visible portion of the lower turbinate, with slight pressure, from behind forward. Shortly after this, a reflex injection on a more or less ischemic basis is shown in healthy people. After a while, the epinephrine effect disappears. Ischemia and injection vanish. The vessel system in these cases reacted with dilation. In certain other cases, a more or less pronounced and sharply confined contraction of the vessels is noticed, the so-called white streak picture. Sometimes it appears immediately; sometimes only after a few minutes. At other times a hyperemia appears first, which turns into the white streak picture. A turbinate is watched for one or two minutes, then after three to five minutes, and, if necessary, also after ten minutes and longer. It can happen that the stroke with the probe remains without reaction after spraying. It can be assumed that the diffuse increased ischemia expresses a hypertonia of the sympathetic system. Rarely it may happen that an injection of the anterior portion of the lower turbinate occurs, even if the rest of the turbinate is ischemic or a white streak picture is present. Probably abnormal conditions are present in this instance, which, however, do not detract from the importance of a white streak.

CONTRIBUTION TO THE ROENTGEN DIAGNOSIS OF THE FRONTAL SINUS. HELMUTH RICHTER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:456 (April 27) 1926.

Contrary to clinical and surgical observations, the frontal sinuses seemed to be missing on one or both sides in occipitofrontal roentgenograms. Frequently, instead of the frontal sinus, or next to very small frontal sinuses, compact bone and exostoses were observed in the orbital cavities. The questionable absence of the frontal sinuses in the first case could be shown as a deception either by the occipitomental exposure (Passow and Graupner), or in all cases of very low, slitlike frontal sinuses by the nasofrontal tangential exposure (Richter). Jansen had called attention to the fact that such flat frontal sinuses furnish unreliable pictures by occipitofrontal exposures. He also mentioned the substitution of the frontal sinus by compact bone, and that its shadow is stronger than that of an inflamed frontal sinus. The author studied 800 cases, 373 female and 427 male, concerning the size of the frontal sinus and the presence of compact bone and exostoses in frontal sinuses of various sizes and of missing frontal sinuses. If the frontal sinuses are classified as large, medium sized, small and very small, the male sex predominates at the beginning, the female sex at the end of the classification. The frontal sinuses were more frequently missing in the female. The author concludes that:

1. Large frontal sinuses are more frequently found in the male (63.64 per cent); small in the female (62.5 per cent).

2. The frontal sinuses are more frequently missing in the female, either on one or on both sides (55.18 per cent) than in the male (44.82 per cent).

3. Large and medium sized frontal sinuses are percentally very rare; small and very small frequently in the female (55.11 per cent), and more rare in the male (17.93) in connection with exostosis formation or compact bone.

4. If the frontal sinuses are absent, compact bone or exostoses are percentally far more frequently found in the female (42.28 per cent) than in the male (13.79 per cent).

5. The presence of frontal sinuses excluded compact bone or exostoses in 80.05 per cent; the absence of frontal sinuses and compact bone and exostoses coincided in 62.07 per cent.

It is known that large frontal sinuses are more frequent in the male than in the female. The unilateral or bilateral absence of the frontal sinuses is perhaps caused by the increased growth of compact bone in these cases. For unknown reasons the bone develops into exostoses. Therefore, it can be explained that compact bone or exostoses are present if the frontal sinuses are missing, or are small or very small. As the latter group is found especially in the female, it was observed, especially in women, in 16.75 per cent of all 800 cases, and in 72.83 per cent of all cases with compact bone and exostoses. In such cases the orbital cells of the ethmoids can reach very far upward, and they must not be taken for very small frontal sinuses. To establish the absence of the latter, the occipitofrontal exposure must be supplemented by an occipitomental, nasofrontal or also by a bitemporal exposure.

AMBERG, Detroit.

Miscellaneous

RHEUMATIC AFFECTIONS. SOME OBSERVATIONS ON THEIR TREATMENT. J. LESLIE ORR, Brit. M. J. **1**:782 (May) 1926.

In acute rheumatic fever the majority of patients progress favorably with rest in bed between blankets, and taking salicylates. But in those who do not respond to do these measures, cultures should be taken from the throat, the teeth and from the fluid withdrawn from any swollen joint. The author has found a long type of streptococcus from which an autogenous vaccine was made. There was a reaction after the first dose, but marked improvement resulted within two weeks. The early use of an autogenous vaccine reduces the tendency to cardiac and other complications.

METHOD OF TREATING ASTHMA BY RADIATION. S. GILBERT SCOTT, Brit. M. J. **1**:939 (June) 1926.

In attempting efficient irradiation over large areas, really a roentgen-ray bath, for carcinoma, it was found that some of the patients who were subject to asthma breathed more freely and remarked that they had lost that tight feeling in the chest. This irradiation was then tried on confirmed asthmatic subjects over a series of twenty-one cases. There was very marked improvement in all cases. This is just a brief statement of the facts observed. The author is not prepared to say in what way the irradiation exerts a beneficial result, but merely suggests that the method might be tried by others.

DIVERTICULA OF THE OESOPHAGUS. JOHN MORLEY, Brit. M. J. **1**:981 (June) 1926.

Three cases of esophageal diverticula are reported in detail, with the operation for removal. The pouch originates in the posterior pharyngeal wall between the transverse and oblique fibers of the cricopharyngeus muscle. There is no evidence of a congenital origin. It is due to increased intrapharyngeal tension. According to Durr, half the cases are associated with goiter. Spasmodic or fibrous stricture of the esophagus may also cause the condition. It is most common in old men. The main point of diagnosis is the regurgitation of undigested food after some hours or days, together with a gurgling sound and fulness of the neck. There is also marked choking and coughing. The only curative treatment is surgical—excision of the sac.

A Preliminary Report on the Radiographic Exploration of the Broncho-Pulmonary System by Means of Lipiodol. John Guy, Edinburgh M. J. **33**:269 (May) 1926.

Of the various methods of administering lipiodol for diagnosis of bronchopulmonary conditions, the author favors the infraglottic or intercricothyroid route. The French school advise a preliminary injection of morphine, but the author does not find it necessary. The patient is placed in the recumbent position, with the head bent back to render the trachea more prominent. Using a 2 cc. syringe filled with a 2 per cent cocaine solution, the skin, subcutaneous tissues and the submucosa of the trachea are very lightly infiltrated then the remainder of the cocaine is injected drop by drop into the lumen of the trachea. All movement of the patient and of the syringe must be avoided, and a very stout or strong needle should be used. After a few minutes the lipiodol is injected from a 20 cc. syringe, using one of the several especially devised needles. The lipiodol should be heated in a water bath up to 50 or 60 C. The amount of the lipiodol will vary with the age of the patient. Some advise inhalations of oxygen during administration. The patient should be so placed as to have the part of the lung to be examined the most dependent. The photograph should be taken within the first two or three minutes after injection. If care is exercised, there will be very few complications. Injury of the tracheal mucosa or injection of the lipiodol into the peritracheal tissues are the most common accidents, and these are not serious.

Stable Nervous Control in Relation to Flying Efficiency. Martin Flack, Lancet **2**:748 (Oct. 10) 1925.

In the selection of personnel for aviation duties, particular attention must be paid to stability of nervous control. In respect to vision, there must be a perfect balance of bifocal vision. Any unstable control of the eye muscles tends to erratic landing. Some experiments are quoted to show that the pilot depends for his sense of position on muscle and joint sensations. Tests for muscular coordination are carried out in an apparatus which records the time taken to bring the control stick and rudder into a certain position in response to a definite stimulus, for instance, a visual stimulus. The effect of distraction is also determined by sounding a klaxon unexpectedly during the experiment. Physical endurance, which is necessary for flights of long duration or at high altitudes, is determined by studying the respiratory and circulatory mechanism. Certain tests are described for each. It has been found that subjects who do not respond well to these various tests have been reported by the instructors as unfit for flying. The degree of higher psychic control is mostly decided by taking a careful previous history, and by carefully observing the subject during the examination. In conclusion, the author points out that it is important to distinguish between states producing temporary lack of nervous stability and control, and states in which such lack is of a more permanent nature.

A Study of Some Aspects of Miners' Nystagmus. G. H. Pooley, Lancet **1**:751 (April) 1926.

The author is a member of the miners' nystagmus committee of the Medical Research Council. He reviews the literature on miners' nystagmus and divides it into two groups, from 1870 to 1907, and from 1907 up to the present. The incidence of miners' nystagmus for the first period, together with the incidence

of incapacity, was the same for Britain, France, Belgium and Germany. Roughly, about 50 per cent of all miners had gross oscillations of the eyes. The incidence is greater in safety lamp pits than in open-lamp pits. In the old days, miners seldom stopped work and when they did they were able to return to work in a few weeks or months. In Britain since the war, the true incidence of the disease is less if anything, but the claims for compensation for incapacity are more numerous than they were in the first period and than they are at present on the Continent. The cause of the disease is considered to be faulty illumination, and according to Haldane the carbon dioxide content of the coal gas is not a factor in the etiology of the disease. The author conducted a research on a series of 100 cases of miners' nystagmus to observe the effect of different strengths of light on the eyes. He found that when the illumination is below a certain strength, there is an oscillatory movement of the eyes. He explains this by pointing out that below 0.002 candle power the central area of the macula loses its supremacy, and that the visual field now most sensitive is a signet ring shaped area surrounding the macula. The feebler the illumination the greater is the difficulty in maintaining strict fixation.

McNALLY, Montreal.

INJURY OF OPTIC NERVE. HALLE, Monatschr. f. Ohrenh. **3**:219 (March) 1926.

In three patients suddenly becoming blind after intranasal ethmosphenoid operations, Halle has performed an external operation into the orbit. He assumed the blindness was due to emphysema, hemorrhage or edema spreading into the orbit and injuriously compressing the optic nerve and its central artery. The retina is destroyed if the blood supply is cut off for more than ten minutes.

Vision did not return in any of the patients operated on mentioned in the foregoing. Halle believes, however, that it could have been preserved if operation was consented to when he suggested, that is, immediately after the sudden blindness ensued.

GLOWACKI, Detroit.

THE LOCAL DIAGNOSTIC VALUE OF THE WASSERMANN REACTION IN THE CEREBROSPINAL FLUID, ESPECIALLY IN CASES IN WHICH A DIFFERENTIAL DIAGNOSIS IS DIFFICULT IN QUESTIONABLE SYPHILITIC OR GENERAL INFLAMMATORY DISEASE OF THE CENTRAL NERVOUS SYSTEM AND ITS MEMBRANES. JOHANNES ZANGE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:449 (April 27) 1926.

Lately, the local diagnostic value of the Wassermann reaction in the spinal fluid has considerably lost in its estimation. It is doubted more or less when an increased permeability of the "meninges" (better, of the choroidal plexus, ependyma and meninges) for hemolysin, and if the occasion presents itself, also for the complement (positive Weil-Kafka's hemolysin reaction and complement proof, Sachs and others), or, when an increased permeability must be assumed, by virtue of an increased permeability or a more pronounced granulocytosis or a shifting to the right of the colloidal gold curve (meningitis curve C. Lange). Zange reports a case in point: A man, aged 56, had latent syphilis without knowing it and without having ever been treated for syphilis. The Wassermann reaction of the blood was positive. Necropsy, later, showed a mesaortitis syphilitica. On admission, the patient's sensorium was very cloudy, he had high fever and suffered from an acute relapse of a chronic middle ear suppuration on the right side. The operation revealed a temporal lobe abscess

the size of a hen's egg. The patient died after ten days. The cerebrospinal fluid was constantly affected, more so before opening the abscess, then less and changeable, more toward the end. Besides slight increase in pressure (always around 200 mm. of water), pronounced pleocytosis was changingly present (lymphocytes and granulocytes). The total protein (according to Brandberg-Zalociecki) varied between 0.2 and 1.0 per thousand, with mostly positive Pandy's reaction and not a characteristic colloidal gold reaction, chiefly normal. Only a day before the patient died, a double curve with two maxima was present, one with the weaker one with the stronger dilution. On the second day after admission, 0.2 per thousand protein (according to Brandberg) was found, a pleocytosis of 20 granulocytes and 136 lymphocytes in the cubic millimeter. A slight quantity of blood was also found (350 erythrocytes in the cubic millimeter). This was not present on the day of admission or on the following day. There was a pronounced hemolysin reaction (lysis without complement addition, still with 1.4 cc. fluid with the original method). The Wassermann reaction, while positive in the blood, was negative in the cerebrospinal fluid, also with 1.0 cc. fluid. In spite of the positive Wassermann reaction of the blood, and in spite of pronounced increased permeability of the liquor sac for hemolysin and even for the complement (as the positive hemolysin reaction without complement addition proves) and in spite of immediate, if only little, addition of blood to the fluid, the Wassermann reaction in the fluid had remained negative even when the highest usual fluid dosis was used. It cannot be said that a Wassermann reaction cannot be positive if the protein content is so low, because, according to C. Lange, it can be positive only if the protein content of the spinal fluid is considerably higher. Weigeldt and Zange have shown that the Wassermann reaction can be positive even if the protein content is as low as 0.2 and 0.25 per thousand. Although one observation does not permit a final judgment, it is very probable that the Wassermann reaction in the spinal fluid has a value concerning local diagnosis. The cooperation of many is needed to elucidate the subject matter. Otorhinologists see cases of meningitis and brain abscesses, especially secondary ones, more frequently than other physicians, therefore, they are especially called on to cooperate.

AMBERG, Detroit.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

March 1, 1926

HARRY L. POLLOCK, M.D., *President, in the Chair*

TEMPORAL LOBE ABSCESS. Presented by DR. SAMUEL SALINGER.

A boy, aged 14, came into the hospital January 13, apparently very ill. He gave a history of headaches and projectile vomiting, which had persisted for several months. He had had a radical mastoid operation nine months previously. Examination per the meatus revealed the mastoid cavity filled with what appeared to be cholesteatomatous tissue which it was impossible to remove. Papilledema was present on both sides, and there was marked contraction of both fields for form and color. There was no focal symptoms except hallucinations of smell; the patient said he seemed to be smelling ether continuously. There was a strongly positive Oppenheim reaction and a suggestive Babinski reflex of the contralateral side.

The old mastoid wound was opened, and it was found that what was thought to be a cholesteatoma was brain tissue which had collapsed into the radical cavity. Later, it was found that the dura had been exposed and incised at the previous operation. The necrotic brain tissue was followed inward and forward, and at a depth of one and a half inches an abscess was encountered. Three ounces of pus escaped under pressure. A tube was placed in the cavity, but slipped out about a week later and the boy had a recurrence of his symptoms. The tube was replaced, but again slipped out, and on this occasion there was great difficulty in getting it back because the capsule of the abscess was very dense and offered resistance to the penetrating tube.

The tube had been in place continuously and the cavity was still draining moderately. Frequent irrigations were necessary to keep the tube open. The boy was up and around and had no complaint except an occasional subjective smell of ether. They anticipated some difficulty in collapsing the cavity, and thought there would doubtless be a discharging fistula until this was accomplished.

DISCUSSION

DR. JOSEPH BECK said that many men who had treated these abscesses had been troubled by having the tube slip out, and called attention to the plan used by an associate of Dr. Mosher of Boston. This physician had reported fifteen cases in which they had used a funnel made of wire mesh, which was fastened to the surrounding skin. As the granulation tissue formed around the funnel it was gradually removed.

DR. EDWIN MCGINNIS said he believed Dr. Mosher received this idea from the British Expeditionary Forces. They used this method of drainage, leaving the funnel in for six weeks and then letting it come out by itself.

DR. SALINGER (closing) thanked Dr. Beck for his suggestion, but thought it would not be feasible at the present time to change the method of drainage, at least until the fistulous tract leading into the cavity was well walled off.

A CASE OF LARYNGECTOMY. Presented by DR. C. F. YERGER.

A man, aged 46, entered the Cook County Hospital, Jan. 28, 1926, because of embarrassed respiration, which was due to partial closure of the respiratory tract.

An emergency tracheotomy was performed, and on further examination by indirect laryngoscopy a growth was discovered that involved one-half of the larynx. At about this time Dr. Pollock presented a patient before the Society who had had a somewhat similar condition, and the removed specimen showed that the carcinomatous growth had extended to the other side subglottically. Dr. Yerger's patient was thought to be a good subject for laryngectomy, and when the larynx was removed it was found that the growth involved both sides as well as the first ring, and possibly the second of the trachea below the cricoid cartilage.

The laryngectomy was performed February 8, in the usual manner, and the incision had completely healed. He had not thought it wise to remove any of the trachea, because of the possibility of tension on the skin, and the trachea was sutured in situ, with the idea of treating the carcinomatous area later by deep roentgen-ray therapy or radium. The operation was performed under nerve blocking anesthesia, according to the method of M. L. Harris. The anesthesia was complete and Dr. Yerger recommended the method very highly, as being better than the infiltration with procaine. This method consists in making the posterior injection at the posterior border of the sternocleidomastoid muscle just a little above the middle, being careful not to let the needle get into the external jugular vein. When sure that no blood has been secured in the syringe, the solution is injected below the deep fascia which reaches the superficial cervical plexus and causes anesthesia of the neck. The opposite side is treated in the same manner. The superior laryngeal nerve is blocked very readily by locating the hyoid, and injecting about 10 cc. of procaine superficially to infiltrate the superior laryngeal. When the superior laryngeal nerves and plexus are blocked, one can proceed without infiltrating the line of incision. There is no pain except in cutting the mucosa of the pharynx, and this can be avoided by injecting the mucosa before incising the pharynx.

A catheter was placed through the nose following the operation. A mistake had been made in not cutting off the closed tip at the end, as one week later, considerable difficulty was experienced in getting the liquid diet through because of obstruction in the lateral opening by accumulated food.

DISCUSSION

Dr. G. W. Boot congratulated Dr. Yerger on the outcome in his patient, because he still had a fistula present in a patient which had been operated on a month before, but he thought the patient would be cured of the carcinoma.

Dr. Alfred Lewy asked Dr. Boot if the recurrent growth in the larynx had been examined histologically.

Dr. Boot replied that it had not as yet.

Dr. Otto Stein said he had two patients on whom he had performed a laryngofissure for carcinoma, with subsequent apparent recurrence, but he thought these recurrent growths were sometimes nothing but granulomas, or healing tissue. Both of his patients had survived for several years. He thought Dr. Lewy's question was very pertinent, and asked Dr. Yerger if there was any extralaryngeal glandular involvement, and whether any glands were excised at the time of operation. He agreed with Dr. Yerger that a local anesthetic was ideal in these cases.

Dr. Salinger stated that the difficulty of recognizing the extent of the carcinomatous involvement below the glottis had been discussed by many, particularly by Pancoast and Makenty. Indirect laryngoscopy, and frequently even direct laryngoscopy will fail to disclose wide subglottic involvement unless the vocal cords are separated to permit direct inspection.

Dr. Joseph C. Beck thought Dr. Yerger's patient, who had given such excellent results up to the present, was in danger of a recurrence if Dr. Yerger

pinned his faith to the roentgen ray and radium. He recommended instead that electrocoagulation be used for destroying the growth.

Dr. Beck had just performed a second operation on the patient whom he presented at the February meeting. Two weeks before he had performed a complete laryngofissure with the aid of the radio knife, the operation being done very rapidly and easily. He made a gross excision, exposed the larynx, cutting the cartilage and everything except a piece of tissue for microscopic examination, with his knife. He wished to have a piece of tissue for examination that would not be affected by the heat from the radio knife. The growth proved to be malignant, so two weeks later he opened the larynx, which was found to be involved on both sides, with no involvement of the glandular structure outside.

He thought the point raised by Dr Stein was quite important. The low glands are much more important than those above, and in those cases not much results are to be expected from the roentgen ray or radium.

Dr. Boot said that as the trachea is very movable, and practically all of it can be excised when necessary, he thought that in this case Dr. Yerger would have done better if he had taken off the upper ring or two that was involved at the time of the operation.

Dr. Harry L. Pollock presented a Makenty apparatus of the type being worn by the patient who was presented at the February meeting, and said that four of their patients are now wearing these, with successful results. He congratulated both Dr. Yerger and Dr. Boot on their laryngectomy. In regard to laryngofissure, he recalled a patient who was presented in 1925 who had a growth that was localized on one side. He took away the cricoid cartilage and removed the growth. The wound healed very rapidly, but at the end of three or four months there was an apparent recurrence. He delayed doing anything for a time, thinking the growth was a granuloma or a large polyp, but as it increased in size he removed a piece by laryngoscopy and then performed a laryngectomy. He considered this a good method of procedure when the growth is localized to the region of the cord. By direct laryngoscopy, and by separating the cords, one can obtain a fairly good idea of the extent of the growth, and it can be removed by window resection or by laryngectomy. He had performed this operation on four or five patients who were alive after five years. He called attention to the recent report of a southern physician who performed a laryngectomy for a supposed carcinoma of the larynx, without biopsy, and later found the larynx was tuberculous. Dr. Pollack believed there was no danger in making a biopsy, and that it made the operation much safer.

Dr. Yerger (closing), in reply to Dr. Stein, said that he was surprised not to find any lymphatic involvement in such an extensive growth, and no glands were removed. The growth looked typical of carcinoma and no biopsy was made. The Wassermann reaction was negative; the sputum and roentgenogram of the chest were negative for tuberculosis, and he was convinced that the diagnosis of carcinoma was correct.

For feeding, he thought the Rehfuss duodenal tube through the nose was much better than the ordinary catheter.

In addition to the local anesthetic they gave ¼ grain of morphine and 1/150 grain scopolamine one hour before the operation.

Unusual Eye and Systemic Complications of Nasal Obstruction. Presented by Dr. G. J. Musgrave.

The first case reported was that of a man, aged 32, who had first consulted Dr. Musgrave about three years prior to this regarding the correction of a

divergent strabismus. He gave a history of having been "shell shocked" during the war. He was in bed for a week, and following that had diplopia with divergent strabismus. On examination, he found normal vision and no ocular paralysis. To complete the examination for contributory causes, he placed a pledget of cotton saturated with a 10 per cent solution of cocaine at the anterior portion of the middle turbinate bone, on account of a deviated septum. To his amazement the eye became straight and had remained straight up to the present, with no other therapeutic measures.

The second case was that of a woman, aged 28, who gave a history of an epileptic attack at the first menstrual period, and the attacks had persisted since that time. She passed through the usual treatment of internists, but, two years before coming to Chicago, nausea, vomiting and severe suboccipital headaches developed. Because of these symptoms she was referred to Dr. B. W. Sippy, who made a thorough examination, and decided that the same influence that caused the headache and convulsions caused the gastric symptoms. She was referred to Dr. Frank Allport because of a convergent strabismus, and was later referred to Dr. Musgrave for a tonsillectomy. She complained of having lost her sense of smell. These was some evidence of sphenoidal and ethmoidal trouble, and a complete exenteration was performed. With the first touch of cocaine the patient went into an epileptic convulsion, so the anesthetic was immediately changed to a general one. She made a complete recovery, had had no further convulsions, and the convergent strabismus had disappeared. Three years had passed since the operation.

The third patient had been operated on for a mastoid infection, and was presented to emphasize the fact that such operation is sometimes required, even when the typical symptoms of mastoid abscess are lacking. Dr. Musgrave had seen many patients go on to complete deafness because the attending physician refused to operate for otorrhea without the classical mastoid symptoms. This patient's hearing was restored to normal, and he had no further trouble following operation, the sole indication being a persistent bilateral otorrhea with deafness nine months after scarlet fever.

DISCUSSION

Dr. A. A. Hayden said that he had recently been informed that there was no mastoid work done at the hospital for contagious diseases. The physician who told him this concluded that as all the patients recovered from the contagious disorder, or whatever caused the mastoid infection, there were many unnecessary mastoid operations performed in Chicago.

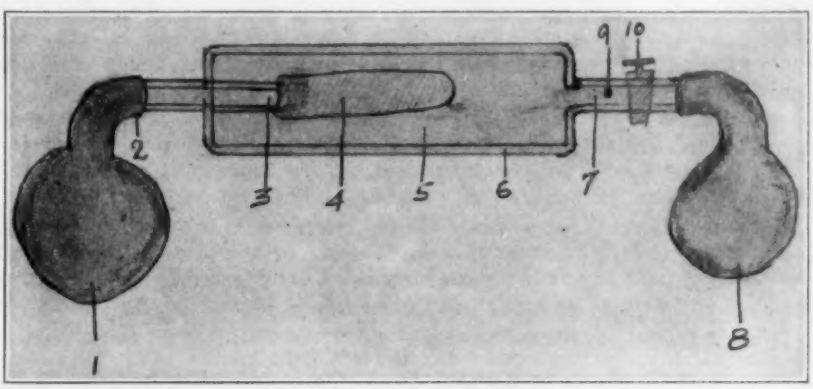
Dr. G. W. Boot said he had recently been asked to have a patient admitted into the County Hospital who had just been discharged from quarantine for scarlet fever, had developed a mastoiditis, and whom private hospitals refused to admit. He also said there was great difficulty in getting any patient with scarlet fever with a mastoid infection into a private hospital, and he thought some provision should be made by which this could be done.

Dr. Robert Sonnenschein believed some of the hospitals went on the principle that exclusion is not necessary after the quarantine period of scarlatina has ended, provided streptococci are not found in the secretion. Recently, he had a case which he took up with the superintendent of the Michael Reese Hospital, and, after consultation with the attending physicians, it was decided that there was no danger in admitting the patient. The child was removed to the Sarah Morris Memorial Hospital. Dr. Sonnenschein was convinced that there was no danger, when the quarantine had been raised, in admitting these patients to the hospitals.

FOREIGN BODIES IN THE ACCESSORY NASAL SINUSES. ABSTRACT OF ENTRANCE THESIS. Presented by DR. M. BRYED WILSON.

The essayist divided the cases in the literature since 1890 into five groups, according to the nature of the foreign bodies, and reported one case of infection of the maxillary sinus which was shown to be due to the presence in the sinus of a pipe stem. Group 1 consisted of foreign bodies of dental origin, of which seventeen observers had reported nineteen cases. Group 2 consisted of calculi, of which two observers had reported two cases. Group 3 consisted of instruments and appliances, of which thirty-three observers had reported thirty-five cases. Group 4 comprised gun pieces and bullets, of which forty observers had reported forty-one cases, and group 5 comprised miscellaneous foreign bodies, of which twelve observers had reported thirteen cases.

Dr. Wilson stressed the unquestionable value of the skiagram in the diagnosis of nasal sinus disease. The reported cases showed that a foreign body in some instances may be tolerated in a sinus for many years without producing suppuration. In some cases, facial neuralgic pains were almost the only symptoms. There was an apparent preference for the Caldwell-Luc type of operation in these cases.



The diagram represents a system of tubes filled with water. *1* is a compressible rubber bulb, the diagrammatic equivalent of the source of the vascular pulsation, namely, the heart, *2, 3, 4,* the vascular system of the labyrinth; *4,* is a flexible rubber finger cot; *5, 6* and *7,* a glass container, the diagrammatic equivalent of the labyrinth and its endolymph content; *8* is a flexible rubber bag and tube corresponding to the saccus endolymphaticus; *9* is a small metal indicator suspended free in the fluid, the diagrammatic equivalent of the crista ampullaris; *10* is a glass stop cock.

Small compressions of the bag (1) are communicated to the fluid (5) through (4) and set in motion indicator (9) when the stop cock is open and the bag (8) (saccus endolymphaticus) permits the pulsations. When the stop cock is closed, compressions of bag (1) have no effect on the indicator (9). Illustrating the effect produced by Portmann in blocking off the ductus endolymphaticus.

RETROPHARYNGEAL ABSCESS WITH EROSION OF NORMALLY PLACED INTERNAL CAROTID. Presented by DR. T. C. GALLOWAY.

The patient, a girl, aged 11 years, had been ill six days when first seen in consultation February 15, and was complaining of pain on the left side of the throat referred to the ear, with some difficulty in swallowing. Behind the left

faucial pillar a beefy swelling about 2 by 3 cm. only slightly raised was seen. When next seen, four days later, there was a rounded pole swelling about 3 by 5 cm. in diameter, which fluctuated. This was opened and about 25 cc. of thick greenish pus was evacuated. There was almost no bleeding. Twelve hours later there was a sudden hemorrhage with the loss of about 500 cc. of blood, which stopped after the nurse applied pressure with cotton. Thromboplastin and fibrogen were given for two days, then calcium lactate by mouth. The patient was removed to the hospital and did fairly well, having a moderately blood stained discharge of pus. Five days later, 50 cc. of blood escaped suddenly. On account of the temperature elevation the wound was gently reopened with forceps three days after the incision. On February 28, Dr. Galloway began to make a more lateral opening with scissors, but desisted at the resistant feel of the swelling.

At 10: 30 p. m., February 28, again suddenly, between 300 to 500 cc. of blood was lost. The patient was very weak and almost pulseless. In the morning, 100 cc. of blood had been given intramuscularly, but the patient was taken to the operating room and given 450 cc. of uncitrated blood by the syringe method. A throat and neck set were ready. As the last syringe was emptied the patient spat out a mouthful of blood, and on looking at the throat a stream of blood about 15 mm. wide could be seen. Pressure and packing did not control it, nor did pressure of the carotid against the carotid tubercle by an assistant. Just after ligating the internal carotid the patient stopped breathing from exsanguination and aspiration of the blood, which the Beck suction apparatus was unable to remove fast enough.

At necropsy, Dr. J. Lisle Williams found that bleeding from this region would have to come from (1) The ascending pharyngeal, a rather small artery; (2) anomalous branches of the external carotid, or (3) from erosion of the internal carotid, which has no branches in its cervical portion.

From the history of this case and the one presented by Dr. Boot in February, and from the consideration of the blood supply of this region, it seemed to Dr. Galloway that any sudden and profuse hemorrhage associated with retropharyngeal abscess is an indication for immediate ligation of the internal or common carotid.

The necropsy report was as follows: When the tissues of the neck were dissected free, an abscess cavity filled with blood and purulent material opened. It lay behind and to the left of the esophagus, extended downward to the level of the roof of the thorax, and contained necrotic material, fluid and clotted blood. The arterial wall of this abscess cavity extended backward and upward on the left side, where it was in contact with the bony structure of the pharynx, and here it was eroded through the wall of the internal carotid artery. The channel of the vessel at this point took a sudden turn as it entered the base of the skull cavity, with a bulge inward. The aperture through which the hemorrhage came was apparently 3 mm. in the greatest diameter.

DISCUSSION

Dr. Otto Stein believed these cases were not very uncommon, and thought that Dr. Galloway's last remarks should be kept in mind by everyone. When such a large amount of blood was lost in connection with a laryngeal abscess, one should make an immediate ligation of the internal carotid artery.

Dr. Galloway (closing) stated that statistics showed that the operative mortality of ligation of the internal or common carotid is between 2 and 3 per cent, and cerebral softening or other severe damage occurs in about twenty-five

per cent. With the poor circulation and pronounced anemia there would have been great danger from ligation in his patient, but he thought perhaps this should have been done earlier.

CRANIAL INJURY FOLLOWED BY INVOLVEMENT OF THE LABYRINTH AND COCHLEA. Presented by DR. C. F. YERGER.

A porter, aged 39, who had been married twenty years and had four healthy children, came to the Illinois Eye and Ear Infirmary Dec. 15, 1923, with the history that two days previously he had been struck on the head with a billiard cue. He stated that he was unconscious for a few minutes, which was followed by vertigo, vomiting, disturbed vision and ataxia. There was exudation of bloody serum from the right ear. Deafness in the right ear was noticed immediately after the accident, which had persisted. The vestibular symptoms were present for a period of four hours, with no recurrence since then. Examination of the right membrana tympani disclosed a rupture which was plugged by a blood clot.

Feb. 24, 1926, the man returned to the dispensary on account of deafness and tinnitus involving the right ear. The functional test showed the observations of nerve deafness. The left ear was normal.

March 8, the vestibular tests on rotation with the head erect, showed a nystagmus of 7 seconds' duration when rotated toward the right, and of 9 seconds' duration when rotated toward the left. With the head back, rotation toward the right produced a nystagmus of 9 seconds, and rotation toward the left of 14 seconds. The caloric test was normal on the left, but on the right with the head erect no nystagmus resulted; with the head back there was distinct nystagmus. There was no spontaneous nystagmus or past pointing. The induced past pointing and falling tests were normal. The patient gave a history of a doubtful chancre before marriage, but had had several negative Wassermann reactions.

DISCUSSION

DR. YERGER thought this was undoubtedly a case of skull fracture which occurred when the man was hit on the head with a billiard cue two years ago, when he became unconscious and bled from the nose. He did not know whether it was a basal fracture or not, but the man had involvement of the labyrinth and of the cochlea. He had stuporous symptoms, was very dizzy, nauseated, vomited, and probably had nystagmus. The significant observation was that two years afterward he had complete nerve deafness. There was involvement of the organ of Corti, and the vestibular branch was functioning. He had regained some of the vestibular function, but had lost the cochlear function. About 25 per cent of the patients with basal skull fracture have involvement of the labyrinth, which is due to the hemorrhage into the membranous labyrinth. A sudden, very loud noise will sometimes produce the same symptoms. Dr. Yerger believed that the pars petrosa may have been fractured, in spite of the fact that the man apparently had a slight injury.

UNUSUAL SINUS ROENTGENOGRAM OBTAINED BY ORAL RHINOSCOPY. Presented by DR. A. A. HAYDEN.

The patient was a man, aged 53, with an extensive facial disfigurement. Three years ago a right upper wisdom tooth was removed, with very little difficulty. There was a history of tuberculosis on his father's side of the family, and the patient also admitted having had syphilis thirty years ago, for which he had received treatment. Following the tooth extraction, an induration of the right cheek developed, which persisted for several weeks, when a fistula

opened into the mouth. He then consulted a physician in Waukegan, who collected a specimen from the fistula, sent it to a Chicago laboratory for diagnosis, with the resulting report of carcinoma. The patient then received roentgenotherapy and later, in Chicago, radium was applied. At the expiration of three months he was informed that the treatment had not been effective, and he would have to have a resection of the malignant tissue in his mouth. This was done in two stages, at intervals of about five months. All of his teeth were removed and the upper part of his jaw sloughed out. The patient was satisfied with the result obtained, but was now looking for plastic surgery to repair the disfigurement. He was not Dr. Hayden's patient, and he did not know whether or not the diagnosis of carcinoma was correct.

Dr. Hayden then exhibited pictures, of a woman, aged 30, who consulted him because of disease of the right antrum. The first molar had been extracted several months before; following this a fistula formed, and the patient was greatly distressed because food and liquids trickled through her nose. This case offered an excellent opportunity to use lipiodol in the examination of the sinuses. Dr. Hayden did not know whether this preparation would prove ideal for use in the sinuses, but thought that when it showed a very clear-cut outline of the antrum it might be of value. It could be injected through the eustachian tube and the outline secured in this way.

DISCUSSION

DR. ALFRED LEWY asked if there was any reaction or local irritation from the lipiodol.

DR. JOSEPH C. BECK believed the injection of lipiodol would be of no value unless it was mixed with some substance which would fill out the cavity of the antrum, as it is too thick and oily to do this satisfactorily alone. It has an advantage over bismuth because it is nonirritating, and it has proved of great value in examination of the bronchial tree. He thought if it could be incorporated in petrolatum so that it would fill out the sinus cavities it might be of value.

DR. HAYDEN (closing) said he realized that the antrum cavity was not completely filled out in his case, but had thought this was due to his faulty technic. The needle he used did not fit into the dental root very snugly, and therefore he could not carry enough pressure to cause complete filling of the cavity. It should be injected with a large needle.

PRIMARY OSTEOMYELITIS OF THE MASTOID BONE (TRAUMATIC); REPORT OF CASE AND REVIEW OF LITERATURE. ABSTRACT OF ENTRANCE THESIS. Presented by DR. MYRON E. KAHN.

A white girl, aged 9, was admitted to the hospital, June 14, 1925, complaining of pain and swelling behind the right ear. She gave a history of having been struck behind the right ear by a ball thrown four weeks previously. Pain had been present behind the ear since that time, with many attacks of vertigo and headache. The pain was worse at night and disturbed her sleep. One week before entrance swelling and tenderness developed behind the ear, but there was no pain. The temperature at times had been 104 F. There had been no vomiting. She had had no previous ear trouble. The general examination was negative. The left ear was normal, the whispered voice being heard at 4 meters. The right ear showed a small amount of cerumen and desquamating epithelium. There was some sagging of the posterior wall, and redness and tenderness behind the ear. The whispered voice was heard at 2½ meters. The white blood count was 15,150. Hot applications were advised. On June 20

the white blood count had increased to 17,600, the swelling had become larger, there was definite fluctuation, and the patient was seen by Dr. Kahn. He found the mastoid region very tender. The ear was pushed forward; the drum was dull, but there was no bulging. A diagnosis of mastoiditis was made, and the usual operation was performed. There was a large amount of free pus, the cortex was hard, and there was a small perforation in the region of the antrum. This was enlarged, and the cortex was removed. The bone was soft and all cells were removed. The antral opening was enlarged, and pus and granulation tissue were removed. Culture of the pus showed *Streptococcus hemolyticus.* The child made an uneventful recovery, and was discharged on July 12.

Other instances of mastoiditis following trauma were briefly reviewed, the references in the literature to primary mastoiditis due to trauma or otherwise, were brought up to date. All cases of mastoiditis, in which the middle ear was not involved at any time, were considered primary, regardless of the cause of the mastoid involvement. Most of the reports on traumatic mastoiditis in the literature had simultaneous or secondary middle ear involvement. Some were pure fractures of the mastoid bone. The case reported herewith was a typical suppurative osteomyelitis of the mastoid, with the middle ear apparently intact at all times. Such instances were considered extremely rare.

THE SACCUS ENDOLYMPHATICUS AND ITS RELATION TO LABYRINTHINE MUSCLE TONUS. ABSTRACT OF ENTRANCE THESIS. Presented by DR. HECTOR G. LAREAU.

Despite the many investigations and publications on the subject, there is still a host of unsettled questions in regard to the physiology of the labyrinth. The gross anatomy of this complicated structure is well known, but the physiology, both as regards the whole apparatus and its various components, is not clear. In the living organism, whether the fluids, traversing the various structures, obey hydrodynamic laws is not satisfactorily known, as investigations have generally been made on isolated labyrinths and not in a living subject. Portmann, who has thoroughly studied the saccus endolymphaticus in fishes, mammals and man, has shown that the method of communication is different from that described by Hertwig. This investigator shows that the saccus insensibly narrows into the ductus, and at about one-third of its course widens out again into the sacculus; it is from this that communication is made with the utricle and cochlear canal by the intermediary of two small canaliculi. There is no direct communication of these structures with the ductus. The endolymph apparatus may be considered as a bilobated structure; one extremity, the saccus endolymphaticus, being intracranial and the other extremity, the sacculus, being the vestibular expansion. The saccus endolymphaticus is embedded in the endolymphatic fossa, which is situated at the level of the external third of the posterior face of the petrous bone. This fossa is quite roomy enough to accommodate variations in the volume of the saccus. The sacculus and saccus endolymphaticus with the duct form an organ which is independent of but in communication with the organ of equilibrium (utricle) and the organ of audition (cochlea).

He briefly indicated the several theories in regard to the excitation of labyrinthine tonus; Breuer's assumption of endolymph currents; Ewald's assumption that the hair cells of the crista were in a state of ciliary motion which constituted a constant stimulus of the hair cells; the theory of otolith pressure on the macula and relative tension differences as propounded by Maxwell and others; and Shambaugh's theory of vascular pulsation transmitted

to the endolymph. Shambaugh's vascular theory appealed to him to be the most logical as it is based on a constantly present physiologic factor, i. e., arterial pulsation. Recent experimental investigation by Wodak and Fisher, from which these authors deduce that tonic vestibular reactions are independent of a vascular reaction, did not appear to him to invalidate Shambaugh's hypothesis. Their artificial vasoconstrictors and vasodilators besides their action on the endolymph have also a certain nervous action.

That endolymph currents exist does not appear to have been seriously denied, although the stimulatory effects of such currents have been debated. One important point appeared to have been overlooked by all investigators, i. e., that it is physically impossible for a fluid to move in a closed tube. Such movements can only take place in a tube when the ends are open and free. The endolymph apparatus or membranous labyrinth in man, for practical purposes, may be regarded as a tube with expansion at each end which allows motion of the fluid contents in either direction. The two expansions are the sacculus and the saccus endolymphaticus. The sacculus may be broadly considered as a necessary and integral part of the labyrinthine apparatus, while the saccus does not seem to be so, unless for the one function of permitting movement of fluid in the labyrinthine organs. This he considered one necessary function of the saccus endolymphaticus: That as an expansion chamber it permits motion of fluids in the semicircular canals and other parts of the labyrinth; were the saccus not there, the labyrinth could be considered as a closed system and movement of the fluid within it would be impossible. The constant to-and-fro movements of the endolymph in the canals, due to vascular pulsations transmitted to it, keep the cristae constantly moving and thus supply the necessary stimulus for labyrinthine tonus. In the absence of such stimuli, there could be no labyrinthine muscle tonus.

A schematic drawing and explanation of the simple experiments performed with an apparatus which was represented, illustrated the physical law which shows the necessity for an expansion sac such as the saccus endolymphaticus in the scheme of the labyrinth.

April 5, 1926

C. F. YERGER, M.D., *Vice President, in the Chair*

INTERPRETATION OF SPEECH SOUNDS BY MEANS OF THEIR TACTUAL CORRELATES. Presented by DR. ROBERT H. GAULT, Professor of Psychology in Northwestern University, on leave with the National Research Council.

The data presented in this paper have been accumulated since the middle of October, 1925, that is, since the paper published in the *Archives of Otolaryngology* **3**:121 (Feb.) 1926, was written.

A curve is presented showing how rapidly a woman, aged 22, congenitally totally deaf (audiometric record 0-0) learned to identify the ten vowel and diphthongal qualities: A (as in ate), E (eat), I (bite), O (boat), U (you), Oi (boil), Ou (bout), Er (refer), Oo (boot), Aw (awe). She began her laboratory exercises on Oct. 16, 1925. Prior to that date she had never been in the laboratory. One session of about forty minutes daily, five days a week, was devoted to this exercise. One other daily session was given up to exercises of a different nature. In the twentieth period of practice on the ten qualities, she made a record of 82 identifications by their tactual criteria alone.

Early in 1926, an improvement in technic made it possible in seven sessions to bring a new and wholly unpracticed subject (a congenitally and totally deaf youth, aged 18) to 72 identifications in 100.

Practice at identification of 120 colloquial sentences furnished another opportunity to test the rapidity of learning. One subject who had had an aggregate of 120 hours of laboratory experience before having begun exercises on the colloquial sentences made a perfect record of identifications when the whole list of sentences had been presented only nine times. Another who had had an aggregate of only 80 hours of laboratory training in a variety of exercises, identified over 90 per cent of these sentences when they had been presented nine times.

The foregoing statements of fact afford some indication of the rapidity with which subjects can learn the tactual correlates of an experimenter's speech. The process is sufficiently rapid to justify the expectation that a totally deaf person may find it practicable to acquire the capacity for reception of speech by teletactile audition.

In the course of practice on identification of sentences, subjects have become acquainted with the movement of speech, and its movement is a source of pleasure to them. They enjoy certain forms of poetic verse. They can indicate fairly well how sentences and paragraphs would be read by a good reader. Such familiarity with language as is indicated in this capacity will undoubtedly, after continued practice and in the long run, make improvement in our subjects themselves in respect to their ability to read silently and also, as lipreaders, to catch the drift of a conversation.

It has been demonstrated four times already that when the *feel* of speech is added to the vision of a speaker's face, the deaf subject's accuracy in interpretation of speech is increased by more than 100 per cent above what is possible for him by way of unaided lipreading. It varies naturally with the accuracy that the subject had previously attained as a reader of lips and facial expression.

The superiority referred to is due, in part, to the fact that there are many homophenous words in the English language that cannot be distinguished by vision of moving lips but that can be discriminated by their feel. Data in support of this statement have been set out briefly in the paper referred to in the foregoing. Acquaintance with movement of speech, its emphasis, accent, rhythm and tempo, all contribute to the advantage of touch lip-reading over lip-reading.

There is a great deal of support in the psychology of normally equipped persons for the hypothesis that, after sufficient training, a subject can dispense with the instrument without sacrificing all the advantage that he found in its use; in other words, that he will be a better unaided lip-reader for having practiced with the instrument.

There is tentative support also for the hypothesis that a subject may be enabled to enjoy music by dint of its feel.

DISCUSSION

Dr. J. Gordon Wilson said Dr. Gault had introduced to us a new field of investigation. It was well to keep in mind that he approached this subject from the standpoint of the psychologist; and rightly so, for it is a psychologic problem. While he does not neglect or underestimate the physical problems which require to be solved, it is the mental problem which dominates and must dominate this investigation. The fundamental question Dr. Gault is endeavoring

to answer is: Can the sense of touch, stimulated by the human voice, arouse in the mind word patterns with intrinsic meanings analogous to those normally conveyed by the voice through the auditory sense? He is not trying to get sound through. He is trying to get to the apperceptive mechanism in the brain touch vibrations of such a nature that they will awaken sensations corresponding to the sensations awakened by sounds. To Dr. Wilson, the realization of the storage of these concepts in memory did not seem so difficult a conception as the one he had just stated.

We all know that movements and attitudes of the lips and mouth can be translated into mental concepts of what the voice is saying, and thus sight can act as a fairly efficient substitute in many cases. It can also frame the lips and mouth in a corresponding manner, and so produce the first step in a serviceable vocalization. Can touch also give mental concepts? If it can, it offers greater potentialities, for touch and hearing are closer in their physical properties than are hearing and sight. No one believes that touch or sight can ever rank as the equivalents of hearing. But if touch can aid in the conveyance of speech vibrations, even if it be only to supplement the assistance which lip reading now gives, or to shorten the period required for training, we shall have advanced beyond the stage we now have attained and have added immeasurable advantages to the deaf, both educational and pleasurable.

In discussing this subject, we must discard the words sound and hearing and allow our thoughts to dwell on what is fundamental and common to both, namely, (1) the external physical property of vibrations, and (2) the possibility of such an education by touch. Can the sense of touch, aided by a physical instrument such as Dr. Gault uses, be made so acute and so discriminating that it can be cultivated or educated to distinguish these fine shades of complex vibrations which go to make up speech? We know what touch can do for the blind, but for the deaf it must be vastly more acute to be useful. It would be a mistake to argue that this cannot be hoped for. The linking up of the cerebral associations of touch and hearing is something new, and he would be hasty who would predict the outcome. Still, when one recalls the complexity of speech vibrations as seen in the graphic curves we all know, and when we are mindful of the elaborate mechanism which nature has laid down in the cochlea to aid the separation of vibrational differences, and contrast these with the relative simplicity of the touch mechanism, we may well doubt if touch can ever be an effective substitute even with an excessive amount of training. But this is a long way from saying that there is no future for this work, that it is an interesting piece of psychologic investigation which offers no practical outcome. It appeared to Dr. Wilson that even in this early stage of the work there were many apparent advantages. He referred to only two of these:

1. He believed it would shorten the time now required to make the deaf proficient in lip reading and make vocalization more effective. Efficient lip reading and vocalization is not only shaping the lips in a particular attitude; it is the vibration of the lips and surrounding structures when in this attitude. Dr. Gault's apparatus will give a realization of what vibration is and the kind required, not otherwise obtainable.

2. It promises advantages in the perception of musical rhythms and all the pleasures associated with these. The association of hearing and the muscular adjustment of the body in the inner ear has possibly a deeper significance than is ordinarily believed. Who can have watched deaf mutes gliding gracefully through a waltz or two-step in perfect time to the varying music without realizing that the tactual sense is subconsciously at work? He had no time

to elaborate on this, but thought it was not a long leap from here to the musical enjoyment of rhythm. In the music of dancing it is the accentuated beat which dominates the dance, and it is the accentuated beat which will best stimulate the tactile sense. One would like to know if deaf mutes can dance such complex dances as the tango or such rhythmic dances as folk dances if in these the beat is sufficiently accentuated or can be made so by Dr. Gault's mechanism. In music, it is the regular rhythm dominated by the beat which is the essential. One might compare it to the swell of the ocean, each swell broken by smaller ripples which serve to magnify its grandeur. At present, this swell can be picked up by the tactual organs. If the smaller ripples can be so magnified by Dr. Gault's mechanism as to be perceived, what potentialities lie here for the enjoyment of music. In this field, Professor Gault had presented fascinating glimpses of new mental activities, with possibilities of assistance to those deprived of one of the most important senses. Otologists are deeply interested in this work, and most desirous of seeing it carried farther. Dr. Wilson hoped he would elaborate what he had outlined, and explore still further the possibilities of touch as an aid to hearing.

F. W. KRANZ, PH.D., Riverbank Laboratories, Geneva, Ill. (by invitation), said that he had been interested in Professor Gault's work ever since he had seen the first notices of the earlier portions of it, some two or three years ago. Hearing in the usual sense, as well as in the sense considered in Professor Gault's work, consists in the detection and differentiation of sound vibrations in the air, from the voice or from other vibratory sources. If the differentiation to be made was merely as to the presence or absence of sound it would be simple, and could be accomplished either by means of the sense of hearing or the sense of touch with the use of available apparatus.

It is sometimes assumed that if a deaf person could have a sound of sufficient intensity to listen to, he could hear and understand, but this is not the case. The reason lies partially in the ear itself. The principles are now known for the construction of instruments to produce sounds as loud as one may desire, as intense, in fact, as the air is capable of transmitting, for the air will not transmit too loud a sound without the wave breaking over in somewhat an analogous fashion to large waves on the ocean. If a very loud sound is presented to an ear which has a deficiency somewhere along in its transmission system, with the idea of getting enough energy through this barrier to activate the mechanism beyond the barrier properly, then the portion of the system traversed before the barrier is reached will be called on to transmit energies far greater than it was designed for, and this will result in distortion of the sound quality similar to the distortion occurring in a vacuum tube amplifier when the input is greater than that for which the vacuum tube was made.

Pure tones differ only in frequency and intensity, and the usual sounds are complex tones which are simply combinations of pure tones. Whether a complex tone is pleasing or not depends on the frequency relations of the various component tones. The ear is capable of detecting sounds of different frequencies from 20 to 20,000 cycles per second. The amplitude of a sound at 2,000 cycles per second which the ear is just capable of perceiving, is about one one-billionth of an inch, which is much smaller than any movement which can be detected by the sense of touch. Changes of 10 per cent in the intensity of sounds of ordinary loudness can be noticed by the ear, as can also changes of about 1 per cent in the pitch. The sense of touch will detect changes in pressure of about 25 per cent.

Probably the differentiations made by the tactile sense in Prof. Gault's work are those of rather gross changes in amplitude. Thus, if a person has his

fingers on Professor Gault's telephone receiver which is being actuated by a 500 cycle current, it does not seem probable that he will follow the position of the diaphragm in its excursions occurring in one five-hundredth of a second, but what will probably be detected will be the changes in what may be called the envelope of the motion, that is, the variations in the maximum positions of deflection of the diaphragm, which variations will probably occupy a time of something of the order of a second rather than one five-hundredth of a second. Sentences would probably be differentiated by the sequences of changes of amplitude, and as a sentence has a longer sequence than a word, the possibilities of variation are greater, so it should be much easier to distinguish between sentences than between words. Single sounds, as the vowel sounds, are even shorter than average words, and so are more difficult to differentiate.

If the diaphragm of the receiver should have a pronounced resonance at a particular frequency, there would be a greater amplitude response for sounds at or near this frequency than at other frequencies, and so something of a pitch differentiation would be possible because of this dependent amplitude effect. However, with the type of receiver used by Professor Gault, the resonance is apparently not at all marked. With a given amount of energy the amplitude is greater for the lower frequencies, and this is probably of some assistance to the observer in his differentiations.

Mr. Kranz thought that a further refinement of Professor Gault's apparatus might be made which would give a spatial differentiation of the received frequencies. If four receivers were used, with the resonant frequencies of the diaphragms suitably separated, and a finger were held on each of the four diaphragms, one would obtain a spatial differentiation of the frequencies which, in combination with the amplitude discriminations now being used, should aid greatly in distinguishing between various sounds or sound combinations. Of course, any number of such different receivers could be used, up to the number of digits available. This should accomplish directly and with much better control what Dr. Goldstein does in having deaf children place their five fingers on a membrane over the end of a megaphone into which he is speaking. There is probably a spatial distribution of the sound energy over the surface of the diaphragm, and this is picked up by the five fingers. A large diaphragm would probably differentiate between sounds by a change in the vibratory pattern over its surface.

He thought that Professor Gault was working principally with semimutes and partially with totally deaf subjects. The semimutes have some remnant of hearing left, and can make some differentiations of sound with the ear. It seemed to Mr. Kranz that for the purpose of teaching these people to imitate normal speech, which he thought was one of the important objects of Professor Gault's work, the use of strong amplification, if necessary, on the ears for auditory stimulation would have some advantages. It would give the rhythm which they got from the tactile sense, and also a little help in identification from their pitch sensitivity, although the quality of the sound as a whole might not be correctly perceived. The totally deaf can, however, only be reached through the sense of touch.

He asked how far Professor Gault had gone with the appreciation of music. This would involve delicate discriminations of both frequencies and amplitudes which seemed rather beyond the possibilities of the sense of touch. Probably a person could be brought to a higher state of enjoyment of the tempo of music through the sense of touch, but it would seem that that would be about all that could be done.

DR. PAUL SABINE, Riverbank Laboratories, Geneva, Ill. (by invitation), stated that he was greatly impressed with the results that Dr. Gault had achieved in his unique experiments. Aside from the scientific significance of these results, what occurred to him most forcibly in listening to Professor Gault's paper was the fundamental difference between the physicist's and the psychologist's method of attack on such a problem. Considering how complex even the simplest vowel sound is from the physical point of view, how minute are the physical differences between the vowel "ah" and the vowel "aw" for example, and how unlikely it is that such fine distinctions could be perceived by so coarse a perceptive mechanism as the sense of touch, the physicist would be apt to throw up his hands and say "it can't be done." If he did find courage to attack the problem at all, he would start with the simplest possible case from the physical point of view, as, for example, pitch discrimination between two pure tones. The psychologist, on the other hand, starts on the broad basis of adapting an organism to its environment. He says to the deaf mute "You have one sense left, the sense of touch. Let us see what we can do in the way of substituting that for the auditory sense of normal individuals." He does not start with the physically simplest case; he starts at the very center of the thing, produces the sense impressions in all their physical complexity and leaves it to the creative faculty of the nervous system and the mind to convert them into the symbols of ideas.

This difference in the method of approach was striking, and he had been tremendously stimulated in his own thinking by the evidence of the results that Professor Gault had achieved. In the light of such work, he felt that it takes considerable courage to say anything is impossible. He agreed with Dr. Wilson that the work both as a psychologic experiment and as a practical means of the alleviation of deafness, was tremendously worth while.

DR. FRANK G. BRUNER, Director of Special Schools (by invitation), expressed his great interest in and admiration for Professor Gault's work. He was not certain that the deaf sensed real sounds through the skin, but rather something similar to what a normal person would experience were he to employ the Morse code on the end of the fingers. He called attention to the fact that the appreciation of one's own voice when speaking is made up of a number of elements, namely, bone conduction, and with it certain motor and tactile experiences as well as auditory. He had been greatly surprised on talking into a dictaphone and then listening to his own voice to find that it did not sound at all natural. He thought it a well known fact that those who are afflicted with speech defects often are not aware of their defects, which is due to the fact that so much of the hearing of one's own voice is made up of a muscular sense and bone conduction, which do not come through the ear. Hence one's own speech defects are not so displeasing to one's self as to others.

They are employing the tactile sound appreciation at the Alexander G. Bell school for the deaf where they are teaching totally deaf children to sing. This is done by having the music score placed on the blackboard before the pupils, while the music is played on the piano with the children standing with both hands resting on the piano. They had been astonished to note how easily the children learn to appreciate and sing simple tunes. He thought this was in line with the experiment on which Professor Gault is working, and that the children were sensing differences through the sense of touch cycle, the different tones having different intensity values.

He called attention to some experiments he had conducted to determine hearing differences of different primitive peoples using a telephonic apparatus.

The amount of energy required for normal hearing is infinitely small, ranging in different normal subjects from 6 to 21 hundred millionths of an erg. To make a difference which could be sensed by the skin, this would have to be multiplied from 10,000 to 20,000 times, and this makes a sound so intense that the air will not conduct it.

Dr. Bruner thought that what the children were appreciating in Professor Gault's work was not a difference in the intensity of the sound, but cycle differences. He was greatly interested in the work from a practical standpoint because of its relation to teaching speech to the deaf in the Chicago schools, of which he has charge. They are able to teach intelligible speech at present to only about one in three deaf children, i. e., develop them to a point at which they can speak and read the speech of others readily. If it were possible to perfect a device by the use of which a larger percentage of the deaf could be taught to speak and understand speech, it would be a wonderful help in their education.

Dr. Bruner asked if the subjects were given two sentences which Professor Gault had mentioned, namely, "I am going to the city" and "He said he was tired," whether he could combine parts of those sentences — "He said he was going to the city," and have them accomplish the feat of understanding.

Dr. Elmer L. Kenyon thought the natural basis of comparison for such work was the development of speech in the normal subject. All persons start life without the ability of speech or of understanding speech. The sounds of speech enter the ear of the child as words and combinations of words, and are transmitted to the brain as corresponding sensations. From the memory of these sensations in the auditory word center, the child essays to play sounds on his own speech machine. In order to do this he must analyze words from sentences and sounds from words. This necessity for culling out of words their constituent sounds is the most difficult part of the process of learning to talk. The child finds his greatest aid in the constant comparison of the results of his own efforts with the memories he holds of the same sounds as produced by others. This process of differentiation requires six or eight years for its completion. It was interesting to compare the time which a child requires for learning to talk with the time which Professor Gault's pupils used in learning the tactile speech. Dr. Gault's pupils, comparatively, had taken hardly more than a month out of the six years required by the subject to learn to talk, and yet had secured splendid results in this short period. Dr. Kenyon thought this was one of the most hopeful indications.

He believed undoubtedly that in the child learning to talk, the stimulation to make the differentiations, is brought about largely through the compulsion of the child to play these sounds on his own speech machine. When children learning to talk fail to develop speech normally, the trouble, speaking generally, does not lie in the auditory word area, nor in the motor word area, nor in the peripheral speech area, but in the intelligence area, in the process of differentiating the sensations which come to the auditory word center. It is the difficulty of word analysis that causes the child's trouble. Dr. Kenyon did not know whether it was possible for an individual to carry the apparatus of Professor Gault around with him, but thought this would be the thing to do if possible, for he would thus get much experience in analyzing the sensations produced by the speech of others, and at the same time could compare such sensations with those produced by his own voice. Thus he might be able to improve his own voice and speech.

In almost everything which the child has to learn in the world, care is taken to teach him, whatever the subject may be. The one sense which occurs

without instruction is the development of speech in the young child. He is given words and sentences with any degree of carelessness, and he has to differentiate the words and then the elementary sounds from the words, and until he knows these sounds and is able to place them rightly in any word, he cannot talk normally. Dr. Kenyon considered this a very unintelligent method, and thought one should give the child individual sounds and words slowly spoken, thus reducing the period of learning to talk by 50 per cent. If children are not learning to talk well they are taught individual sounds and single words separately. He thought it was expecting a great deal for Professor Gault to expect a subject in a short time to differentiate words and sentences before they had learned to differentiate individual sounds. Not only vowels, but consonants also, should be practiced individually.

Dr. Kenyon asked Professor Gault whether there was any hope for the differentiation between whispered sounds by these subjects. Since one-quarter of the sounds of English speech are whispered sounds, if so large a factor of speech cannot be differentiated, the procedure would be greatly handicapped.

Dr. Alfred Lewy asked if Professor Gault thought that people beyond middle age, who were not benefited by the usual mechanical aids to hearing, could be helped in a practical way by his method.

Professor Gault said he could not answer Dr. Lewy's question. He had never worked with such a subject, but from the observation he had made he thought this might be possible. He had a patient who was deafened at the age of 16 who is now 27, and he thought that he progressed more rapidly than the others.

Replying to Dr. Bruner's question, Professor Gault said that in the last academic year they tried to accomplish this. He worked first with more than twenty subjects until they learned to identify well ten sentences made up of monosyllabic words, and then made up a list of ten new sentences using the same words, and found that the subjects learned the second list in approximately half the time necessary for the first. He thought this might be because they had become somewhat acquainted with the movement of speech.

Replying to Dr. Kenyon, he said he had not made any systematic experiments with different consonantal sounds, but knew that his students could feel "F" and "St," and such like sounds, in the instrument, although he did not know how accurately they could distinguish them.

As to the possibility of employing the localization on the skin, he said that the new instrument which the Bell Laboratories are making is on the order of a sound filter, having five different receivers, one for each finger.

Book Reviews

NOTIONS ÉLÉMENTAIRES D'OTO-RHINO-LARYNGOLOGIE À L'USAGE DES PRACTICIENS. By DR. G. LIÉBAULT. Paris: Gaston Doin.

As indicated in the preface, this volume is written especially for the general practitioner. The author publishes fifteen lessons which he has given in his service at the Hôpital de la Glacière, Paris, inspiring himself with the teaching of Professor Moure, of the School of Bordeaux.

In his first conference, he describes acute pharyngitis, and reminds us that there exists a certain variety of benign ambulatory diphtheria in which we observe small whitish discrete streaks on the lateral wall of the pharynx. He is against the local applications of iodine, or other irritating substances, and recommends cauterizations with chloride of zinc 1/30, and alkaline gargles.

From the pathogenic point of view, abscess of the tonsil is found in people who have these glands contracted, and the superior extremity hidden in the velum of the palate. As soon as diagnosed, the abscess must be opened early with the knife or the galvanocautery, without waiting for the purulent collection to be too considerable. To prevent relapses, we should perform tonsillectomy.

Paresthesic troubles of the throat present themselves under the form of hypersensibility or hyposensibility. The patients complain of various troubles of the pharynx which are not in relation with what is observed at the time of the examination. We must convince these nervous people that they have no disease, make them sleep, and prescribe antispasmodics. Mentholated oil in local application can also be of help.

Hypertrophy of the tonsil may be congenital. If the hypertrophy is unilateral and the tonsil harder than usual, we must think of a sarcoma. As treatment, the author rejects with reason discission, and igneous cauterization. Under exceptional circumstances in which we must perform tonsillectomy, he advocates tonsillotomy—with Ruault's forceps, which astonishes me. Neither do I see the necessity of cutting the anterior pillar in certain cases for this little operation.

Hoarseness, aphonia and vocal abuse, all the conditions of the larynx produce hoarseness or aphonia which can be considered as the ultimate degree of hoarseness. People who employ their larynx in a defective way can have vocal abuse, which is due to a trouble of the respiratory mechanism, to a bad function of the glottis, or to a misuse of the resonators.

In nasal obstruction, its causes and its consequences, the author passes in review congenital or acquired atresia, malformations of the septum, proliferation of the mucous membrane and tumors. He points out the importance of good nasal breathing from the point of view of general health. He prefers antiseptic salves to oily instillations, and does not advise cauterizations with the galvanocautery for hypertrophic rhinitis. He advocates removing the part of the turbinated bone which is enlarged.

Nasal hydrorrhea manifests itself under two distinct forms, that is to say, periodic coryza called hay fever, and aperiodic or spasmodic coryza which can be produced by different causes. At first we must render the nasal fossae absolutely normal by appropriate treatment, and then use electric vibratory

massage. When the etiology of purulent coryza seems to be obscure, we must make a careful examination of the sinuses.

In oculo-orbital troubles and headaches of nasal origin, infection of the nasal fossae can produce an orbital abscess, even a thrombophlebitis of the cavernous sinus which is always fatal. The nostrils are also the point of departure of malignant or benignant tumors susceptible of invading the orbit. A simple infection of the sinuses sometimes causes trouble of the optic nerve, which necessitates the opening of these accessory cavities. To reach the sphenoid sinus, the author prefers the endonasal to the endoseptal route.

In epistaxis, certain supplementary hemorrhages of menstruation and certain epistaxis observed in arteriosclerosis must be respected. The other cases may be treated by cauterizations with the galvanocautery when the artery is visible, and by anterior packing if it is impossible to localize the hemorrhage. In exceptional circumstances we must practice posterior and anterior packing at the same time. The cause of the epistaxis should be always sought in order to be able to institute appropriate treatment.

Adenoids are observed at all ages of life. They frequently become infected, and may be the cause of complications of the ears, the bronchi and the digestive tract. Producing obstruction of the superior respiratory area, they bring about troubles of the organism. These tumors must be removed with the curet, and the author prefers giving no anesthetic.

In external otitis, foreign bodies of the meatus and wax, we must diagnose diffuse external otitis, and furunculosis of the ear after a complete examination of the auditory canal, in order that these conditions may not be taken for mastoiditis. The foreign bodies of the meatus should be almost always removed with the syringe, and cerumen should be softened with antiseptic glycerine before its ablation.

Acute otitis media manifests itself nearly always as the result of an infection coming from the rhinopharynx. We should then disinfect the cavum. When there is suppuration, the author recommends injections, and at the last period, drainage with dry strips in the canal.

Chronic suppurative otitis media is sometimes maintained by a lesion of the nose or of the pharynx. Washings are equally indicated for a few weeks, and afterwards gauze strips should be employed. If this treatment fails, the radical mastoid operation should be performed, and consecutive dressings with paraffin to bring about epidermization.

Mastoiditis is observed as a sequence to acute suppurative otitis media, and should be diagnosed as soon as possible, as well as its complications. Contrary to certain aurists who favor ice, the author prefers warm damp dressings on the mastoid region, with naturally the disinfection of the middle ear. If trephining is necessary, it must be large, and in cases of infection of the lateral sinus, the author is not a partisan of the ligature of the internal jugular vein.

After having classified the different varieties of sclerous otitis, the author exposes the etiology and the treatment of atrophic otitis. For this kind of condition, unhappily, therapeutics gives very poor results.

This volume is written in a clear, precise style, and is very agreeable to read; I also advise its perusal to all general practitioners who desire to have concise notions on otorhinolaryngology. As Dr. Liébault, "ancien interne" of the Hospitals of Paris, is a brilliant physician of great experience, and in his lectures he has synthetized the renowned teaching of the School of Bordeaux, even specialists would find a real interest in this book.

CAVERNOUS SINUS THROMBOPHLEBITIS AND ALLIED SEPTIC AND TRAUMATIC LESIONS OF THE BASAL VENOUS SINUSES. A CLINICAL STUDY OF BLOOD STREAM INFECTION. By WELLS P. EAGLETON, M.D. New York: The MacMillan Company, 1926.

This is a work of 196 pages, including a complete bibliography with an authors' and subject index. "This contribution is primarily a study of twenty-five personally observed cases of cavernous sinus thrombophlebitis with twenty-one deaths (twelve necropsy reports) and four recoveries, three of which were verified with necropsy findings." The volume contains a contribution of first-rate importance as regards the diagnosis of this condition but especially as to its surgical treatment.